means for giving more precise prognostic information not currently available by other methods.

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Health problems of anaesthetists and their families in the West Midlands

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Summary and conclusions

A survey of all anaesthetists in the West Midlands region—that is, 10% of all the anaesthetists in England and Wales—showed that one in 10 of their children had been referred to a consultant because of a congenital or non-acquired anomaly. Abortions among anaesthetists' families were also common but more so when the mother was an anaesthetist. The anomalies were concentrated particularly in the central nervous system and musculo-skeletal system, and girls were worst affected. The mean birth weights were below normal, more so when the mothers were anaesthetists. Girls with anomalies were particularly underweight. Other effects observed were unexpected infertility, cancer both in the adults and in the children, and, possibly, impaired intellectual development in the children. Many anaesthetising areas were inadequately ventilated, and scavenging devices despite their inefficiency are recommended as a stopgap measure. The results of the study closely resemble those of other studies with similar high response rates to requests for information.

Introduction

Among postulated hazards to anaesthetists are abortion,¹ underweight children,² congenital disorders and cancer in the children,³ and cancer in the adults.⁴ Dispute on statistical grounds⁵⁻⁷ still exists despite experimental evidence that all inhalational anaesthetics are teratogenic.⁸⁻⁹ Only the risk of abortion in women who work in operating departments is accepted by the DHSS.¹⁰ Because of the disproportionate number of children of anaesthetic colleagues in Birmingham having congenital abnormalities¹¹ I decided to survey all anaesthetists in the West Midlands region.

Subjects and methods

A questionnaire was sent to every anaesthetist in the region asking for details of their obstetric and relevant occupational histories for the past 20 years, their paediatric history, and whether they had had cancer. Birth weights were requested together with information on verification. An anaesthetist was defined as a doctor who spent half or more of his or her working time in anaesthetic practice. I set a limit of 20 years because (a) beyond that time details of obstetric history might be unreliable; (b) inhalational anaesthetic agents of 20 years before were still in common use; and (c) broadly the lifestyle of anaesthetists—their age of marriage and of starting a family—had been reasonably static during the period as compared with before, when it was common to delay establishing a family until late in their career.

The questionnaires were followed by a visit to each anaesthetic department to collect and clarify the completed returns. Any missing returns were followed up with letters, telephone calls, etc. My objective was to obtain the maximum return from the sample, which represented 10% of all anaesthetists in the United Kingdom. At each hospital visit details of the ventilation in every anaesthetising area were obtained. The anaesthetic, recovery, and operating rooms were considered as one area.

Respondents were asked not to document hernias, squints, mental health, trauma or the consequences of trauma including birth trauma, or inflammatory conditions. Diseases with an allergic basis were also excluded. The objective was to identify all non-acquired lesions. All lesions either had an anatomical basis or were diseases with an established familial background. Cases were accepted only if the child had been referred to a consultant physician or surgeon, but all referrals were accepted if the consultant established a diagnosis. Thus several conditions not normally regarded as congenital yet with a known familial background were included—for example, Henoch-Schönlein purpura, muscular dystrophy, and juvenile diabetes. Vowles *et al*¹² used the same criteria (except that hernia was not excluded) in their large survey on congenital abnormalities in the community.

I also thought that if congenital disorders were considered as being only those abnormalities that occur in the developing fetus any contribution from the fathers would be excluded (as it would be exclusive of familial disease). In view of the observations by Kripke *et al*¹³ that low concentrations (20%) of nitrous oxide damage the germ cells of male animals one toxic effect of waste anaesthetics could be to induce random mutations such as occur in nature (and are responsible for the emergence of some familial diseases); alternatively by interfering with genetic make-up the dominance pattern of some recessive genes might be altered. This concept of teratogenicity is much wider than that held by Vessey,⁶ who believes that a specific abnormality must be produced if an agent is a teratogen—that is, the damage must be to the fetus at a particular stage of development. To try to separate this genetic effect from any congenital effect I have classified the results according to which parent was occupationally exposed.

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Results

A total of 314 out of 340 anaesthetists (92.4%) replied to the questionnaire, which is comparable to the maximum response (85%) recorded.³ Of the 83 women, 76 (91.6%) replied, so there was no sex bias (see table I). About half the anaesthetists were trainees; hence there was considerable bias towards younger doctors, and consequently there were many whose obstetric performance had not been tested.

TABLE I—Constitution of total sample and obstetric histories of those who had tried to establish a family in past 20 years (some families were not complete).

	Consultants		Trainees		Others		Total		Grand total
	M	F	M	F	M	F	M	F	
	Total sample								
No of requests	135	27	115	49	7	7	257	83	340
No of replies	124	26	109	44	5	6	238	76	314 (92%)
No tested for fertility	100	9	52	17	3	5	155	31	186
No unexpectedly infertile	3	1	1	1			4	2	6 (3%)
No with history of abortions only	5	1	1	1			6	2	8 (4%)
No with mixed history (abortions + live births)	24	3	8	2	1	3	33	8	41 (22%)
% experiencing difficulty	33	50	20	25	33	60	28	38*	30
No of abortions	41	7	12	5	2	4	55	16	71
No of live births	229	14	93	26	7	9	324†	48†	369†
% of conceptions ending in abortion	15	33	11.4	16	22	30	14.1	25‡	15.7

*Difference between sexes not significant ($P > 0.4$).

†Numbers reduced to eliminate double counting of children both of whose parents were anaesthetists and so are entered under both male and female columns.

‡Significance of difference between sexes $P = 0.15$.

OBSTETRIC PROBLEMS

Thirty per cent of the anaesthetists had problems in begetting their families (table I). Unexpected infertility, defined as failure to conceive after exclusion of specific causes, occurred in 6 (3%) of the 186 anaesthetists tested, abortions only occurred in 8 (4%), and 41 (22%) had a mixed history of abortions and live births. Consultants had a slightly higher abortion rate than junior doctors; the difference, which was significant at the 20% level, was assumed to be a slight age effect.

Among those not occupationally exposed to anaesthetics (control group) 10 out of 102 known conceptions ended in abortion (table II). This rate (9.8%) was comparable to that (11%) among controls in another study.¹⁴ In my series the abortion rate among wives of men occupationally exposed was 15.8%, which differed from the controls at the 10% level. The pooled abortion rate among mothers occupationally exposed, irrespective of the father's occupation, was 29.3%, which was much higher than among the controls ($P < 0.05$). The overall abortion rate when either parent was occupationally exposed (18.2%) also significantly exceeded the control rate ($P < 0.05$).

PAEDIATRIC PROBLEMS

Table II gives the mean birth weights of the children in each group. About half the birth weights were obtained from contemporaneous records but there was no difference ($P = 0.5$) between the verified

TABLE II—Obstetric histories, birth weights of live-born children, and occurrence of tumours in past 20 years according to parental exposure to anaesthetics

	Not exposed (controls)	Exposed to anaesthetics			Total
		Father only	Mother only	Both parents	
<i>Live-born children</i>					
Boys	54	126	7	10	143
Girls	38	110	14*	10	134
Total (pairs of twins)	92 (3†)	236 (2)	21	20 (1)	277 (3)
<i>Children with congenital or developmental abnormalities</i>					
Boys	3	11		2	13
Girls	1	11	1	1	13
Total (% of live births)	4 (4.3)	22 (9.3)	1 (4.8)	3 (15.0)	26 (9.4)
No (% of known conceptions)	10 (9.8)	<i>Abortions</i> 44 (15.7)	11* (34.4)	6* (23.1)	61* (18.0)
<i>Mean birth weights ± SE (g)†</i>					
Unaffected boys	Birth weights 3270 ± 78	3380 ± 57	3010 ± 350	3380 ± 100	
Unaffected girls	No of infants 43	96	7	6	
Affected boys	Birth weights 3310 ± 87	3290 ± 65	3090* ± 100	3000* ± 180	
Affected girls	No of infants 32	86	13	8	
	Birth weights 2880* ± 110	3500 ± 90		2180* ± 80	
	No of infants 3	11		2	
	Birth weights 3100	2930* ± 160	2720	3100	
	No of infants 1	9	1	1	
<i>Tumours</i>					
Type	Acute leukaemia (1 case)†	Ependymoma (1 case); lymphoma (1 case)			2

*Significance of difference from peer control group $P < 0.05$.

†One father had reduced anaesthetic commitment to only occasional sessions at time of conception; mother had no hospital connections at time.

‡Several families were unable to provide children's birth weights, so that numbers recorded are lower than group totals.

Thirty-five anaesthetists' families had been completed more than 20 years before, which reflects the rapid recent growth in the number of anaesthetists. Within the 20 years 186 anaesthetists had tried to establish a family, albeit many had not completed their families. The 35 older anaesthetists had between them 36 children, while the remaining 279 had 369 children. This second group included five families in which both parents were anaesthetists and 12 others in which the spouse had also been occupationally exposed during at least one pregnancy or abortion.

birth weights and those recalled from memory. The data were therefore combined. Some birth weights could not be obtained.

The expected mean birth weight of children born in this social class is $3420 \pm \text{SE } 10 \text{ g}$,¹⁵ but all the mean birth weights of the unaffected children were below this. Children born to exposed mothers weighed 10% less than the expected mean birth weight, which constituted a greater reduction than reported elsewhere (50–70 g).^{3, 16} Their birth weight was also lower than that of children born in the lowest social class¹⁵ and children of mothers who inhale cigarette smoke.¹⁵ Girls

were particularly underweight, and even more so were affected girls, who were 15% below the expected birth weight (table II). In contrast the mean birth weight of affected boys was not reduced.

CONGENITAL ABNORMALITIES

Of the 277 children born to anaesthetists during the 20 years, 26 (9.3%) had a congenital abnormality or developmental problem. This incidence is comparable to that (8.8%) reported elsewhere.³ Three of the children died. Table III lists the diagnoses in rank order according to severity. The rankings are very crude—Was the condition fatal, nearly fatal? etc. A significantly greater proportion of girls had major clinical disorders ($P < 0.05$). This agrees with my reported observation¹¹ and accords with the data on birth weights.

36 children born to older anaesthetists developed multiple minor convulsions and died within a year of birth. No further details are available but the case closely resembles a more recent one.

Case 1—This child was a first-born male. The father was an anaesthetist; the mother had no hospital connection during pregnancy. Labour was complicated by a prolapsed cord requiring immediate caesarean section, but five minutes after birth the Apgar score was 8. At three months the child developed right-sided twitching, then hourly minor fits. Electroencephalography showed a typical hypsarrhythmia with frequent slow waves and right-sided spikes. The fits proved difficult to control and the child's development was extremely slow. The probable diagnosis is West's syndrome, which has a poor prognosis.

A third child born within the survey period had even more severe neurological problems.

Case 2—A second child, a girl, was born to a male anaesthetist (the mother

TABLE III—Diagnoses of congenital or developmental disorders ranked in order of severity

Rank order	Diagnosis	Sex of child	Age at diagnosis (years)	Birth weight (g)	Specific treatment
Children born in past 20 years to parents occupationally exposed to anaesthetics					
1	Exomphalos	M	Birth		None: died
2	Bulbar palsy, talipes	F	Birth	2000	None: died
3	Scoliosis, ependymoma	F	Not stated	2790	Craniotomy: died
4	Aqueduct stenosis	F	6	3120	Craniotomy
5	Bilateral hydronephrosis	M	<1	3520	Ureteric transplantations
6	Henoch-Schönlein purpura	F	3	2980	Drugs
7	Hypothyroidism	F	<1	3180	Drugs
8	Diabetes	F	4		Drugs
9	Scoliosis and CDH	F	<1 and 11		Multiple operations
10	West's syndrome	M	<1	3060	Drugs
11	CDH and hydrocephalus (?*)	F	1	3100	Multiple operations
12	Epilepsy	M	2	2270	Drugs
13	Epilepsy	F	10	2270	Drugs
14	Cleft lip/palate	F	Birth	2750	Repair
15	Cleft lip/palate	F	Birth	3180	Repair
16	Cleft lip/palate	M	Birth	3290	Repair
17	Pulmonary stenosis	F	7	3490	Observation
18	Atrial septal defect	M	19	3400	Observation
19	CDH	F	Birth	3400	Plaster cast
20	CDH (mild)	M	Birth	3300	None
21	CDH (mild)	M	3	4200	None
22	Shortened tarsus	M	<1	2100	Too young
23	Talipes	M	Birth	3690	None
24	Encysted hydrocele of cord	M	2	3350	Operation
25	Undescended testes	M	2	3730	Operation
26	Calcaneovalgus	M	Birth	3340	None
Children born to parents not occupationally exposed to anaesthetics (controls)					
1	Cardiac myopathy	M	16	3120	
2	Scoliosis	F	Not stated		
3	Thyroglossal cyst	M	Not stated	2780	
4	Undescended testes	M	2	2760	
Children born more than 20 years before to parents occupationally exposed to anaesthetics					
1	Exomphalos	M	Birth		Died
2	"Cerebral disorder"	M	<1		Died aged 1 year
3	Clubfoot	Not stated	Birth		Multiple operations
4	Muscular dystrophy	M	10		
5	Undescended testes	M	<2		Orchidopexy

*Interpretation obscured by episode of neonatal meningitis.
CDH = Congenital dysplasia of hip.

Of the 36 children born to older anaesthetists—that is, before the 20 years covered by the survey—five had congenital or developmental problems (table III). Similarly four of the 92 children in the control group (4.3%) had congenital or developmental problems; this incidence was comparable to that in the general population (see table IV).

When the lesions were classified according to type there appeared to be an appreciable excess of disorders of the locomotor system as compared with the general population. This confirms my reported observation¹¹ and agrees with the findings of Corbett *et al.*,³ albeit in their study the musculoskeletal disorders were less severe.

OTHER EFFECTS

Although not asked for, three children were reported as having problems of intellectual development or communication difficulties of the dyslexic pattern severe enough to warrant extensive neurological and otolaryngological investigation. This suggests that there may be further, more subtle changes in addition to the gross neurological disorders that were seen.

Some of the neurological problems were very severe. One of the

had no hospital connection; their first child, a healthy boy, was born before the father took up anaesthetic practice). At birth she was noted to have severe talipes, and there was widespread irregular muscular paresis. At two months visual awareness could not be established. Convulsions developed, then spasticity and feeding difficulties with recurrent aspiration pneumonia from bulbar palsy. She died at the age of 5 months. Necropsy showed some thoracic scoliosis and irregular muscle wasting, particularly of the diaphragm and leg muscles. There was extreme loss of neurones in many areas of the brain—hippocampus, olives, and dentate nucleus—and in the spinal cord. The cerebral convolutions were simplified and there was slight dilatation of the ventricles.

The following case is not included in tables I-V because the parents left the Birmingham region before the survey began.

Case A—Both of the child's parents were anaesthetists. She was conceived about three and a half weeks after they had been away from anaesthetics on holiday. Soon after conception both parents returned to work but the mother stopped at the end of the first trimester because of threatened abortion. The child was born microcephalic and with premature fusion of the cranial sutures. There was gross cleft palate, harelip, and no nose. By the age of 2 weeks heavy medication was needed for severe convulsions. Reconstructive surgery was thought to be inappropriate. She died aged 4½ months from aspiration pneumonia.

The incidences of central nervous system disorders, bony disorders,

and harelip or cleft palate or both among the children of anaesthetists were much higher than in the general population (table IV), yet case A suggests that if chronic exposure to pollutant anaesthetics is a hazard then the effect must persist for an appreciable period after leaving anaesthetic practice.

CANCER

Two of the children developed tumours (table II), an incidence of 1 in 138. A third child developed leukaemia, but at the time of that conception the father was doing only occasional anaesthetic sessions. This incidence is comparable to that observed by Corbett *et al.*,³ who reported three tumours (neuroblastoma, thyroid, and parotid) in two out of 434 children born to women anaesthetists.

Some hospitals had installed scavenging systems but many could not afford to do so. Where scavenging was installed the anaesthetists reported increased well-being, which, perhaps more important, was noted by their wives. One wife could tell invariably whether her husband had or had not worked in a scavenged operating theatre even though the work loads were similar and the principle agent used was nitrous oxide.

Discussion

These anaesthetists in the West Midlands region constituted 10% of all anaesthetists in England and Wales. About one in 10 of their children had a congenital or developmental anomaly. One-third of the anaesthetists who were married had an

TABLE IV—Incidences of congenital abnormalities in present series and in normal population

	Present series		Rate/1000 normal population			
	No of cases	Rate 1000	Devon ¹²	Birmingham ¹⁹	S Wales ²⁰	Liverpool ²¹
No of children at risk:	277		25 432	186 046	92 982	91 176
Central nervous system*	6	22	1.26	1.98	1.16	Not clear
Hip dysplasias	5	18	2.99	0.91	0.85	0.67
Foot or ankle	4	14	7.67	5.93	3.42	2.65
Cleft lip/palate	3	11	1.77	1.98	2.96	1.54
Circulatory	2	7	7.47	4.23	4.38	5.03
All†	26	94	46.6	26.7	35.7	23.9
Significance of difference (P)			0.001	0.0001	0.0001	0.0001

*Excludes meningocele and anencephaly—that is, group 3 of Devon study.¹²

†Excludes squint and hernia but includes all talipes. (NB. Numbers for Birmingham anaesthetists study exclude five other children out of 36 born to older anaesthetists who also showed congenital or developmental abnormalities.)

TABLE V—Provision of some form of ventilation in anaesthetising areas in hospitals in West Midlands region

	No ventilated	No (%) not ventilated	Total
Operating rooms	130	46 (26)	176
Casualty theatres	11	21 (66)	32
X-ray departments	2	29 (94)	31
Maternity theatres	16	11 (41)	27
Total	159	107 (40)	266

Two of the 76 women anaesthetists in my survey developed breast cancer. A third case was reported in the wife of an anaesthetist; the wife, a dentist, regularly gave dental anaesthetics. Six of the 238 male anaesthetists also developed cancer (four skin cancers, one lung cancer, and one rectal cancer, in which there were known familial factors). The number of anaesthetists who developed cancer and retired is unknown.

VENTILATION

There were 266 sites in the West Midlands region where inhalational anaesthetics were regularly given by anaesthetists. Other sites—for example, cottage hospitals where occasional anaesthetics were given by general practitioners, places where electric convulsion therapy was given, and dental surgeries—were excluded. Of the 266 anaesthetising areas, 107 (40%) had no artificial ventilation or air conditioning at all (table V). X-ray departments were particularly poor in this respect. A quarter of all operating theatres had no ventilation.

Forms of ventilation that were present varied from a primitive fan to the sophisticated Charnley air conditioning unit. In only one hospital were regular checks made that the equipment was working to specification, and in that hospital the air was recirculated. Spot checks in two other hospitals showed that the numbers of changes per hour were less than three-quarters of the rated number of changes. In all other hospitals the anaesthetists relied on the engineers, who relied on the ratings of the plant. One theatre with a very high number of air changes suffered a different type of pollution—namely, smell when local farmers were spreading manure.

obstetric problem. Apart from the abortion rate there was little difference between the families of male and female anaesthetists. Their children were born underweight, and girls were particularly affected. Where they overlap these results agree with those of other surveys.^{1-4 11} The most striking agreement is with the findings of Corbett *et al.*,³ who recorded an 8.8% incidence of congenital abnormalities (as compared with 9.3% in this survey), abnormalities of the musculoskeletal system (also found in this survey), and unexpected cancer in children (also found in this survey). The principal difference between the two surveys was that Corbett *et al.* analysed children born to women anaesthetists, whereas in my study male anaesthetists predominated. The return rates in the two surveys were 84.5% and 92.4% respectively.

The high incidence of musculoskeletal abnormalities agrees with experimental evidence.^{8 9} Most such studies have examined prolonged exposure of anaesthetists to doses in the clinical range, yet Healy and Wilcox¹⁷ found that trichloroethylene at the threshold limit (100 ppm) induced fetal abnormalities in animals. There is no evidence of a finite lower limit of anaesthetic gas pollution below which congenital abnormalities are unlikely to occur. Such a study is beyond the resources of most anaesthetic departments.

My results differ in two ways from those of Knill Jones *et al.*,^{14 18} who surveyed all anaesthetists in the United Kingdom. In my study the return rate was 92%, whereas Knill Jones *et al.* had returns from 82% of women and 70% of men. In my study the respondents were asked for all diagnoses (excluding acquired lesions) made by another consultant, whereas Knill Jones *et al.* asked about congenital abnormalities, which could emphasise anatomical anomalies. Thus disorders of function with a known familial background, such as Henoch-Schönlein purpura, muscular dystrophy, and juvenile diabetes, may well have been omitted.

Nevertheless, in my series 21 of the 26 affected children had an established anatomical disorder, which is significant at about the 5% level when compared with the highest incidence of congenital abnormalities found in the normal population (table

IV). The mean incidence of congenital abnormalities in the population at large is difficult to determine^{12 19-21} and varies with the criteria used for diagnosis. I used the same criteria as used by Vowles *et al*,¹² except that I excluded hernia. Such comparisons do not, however, take into account the pronounced excess of severe problems among girls or the distribution pattern of the type of abnormalities observed. Both of these also imply that the observed results are different from those obtained in the normal population and its pattern of congenital problems.

The occurrence rate of primary tumours in this study was 1 in 138 children, and in the study of Corbett *et al* 1 in 145 children. The combined incidence is about 7.0/1000. In contrast the incidence of all tumours in children and young adults (below 20 years) in England and Wales is 11/100 000²²—that is, there was a 60-fold increased incidence among the children born to exposed parents. Among the 76 women anaesthetists in my study 2 (2.6%) had cancer of the breast. Corbett *et al* found 15 cases of breast cancer among 525 women anaesthetists (2.9%). Another survey²³ also disclosed a high incidence of breast cancer among women anaesthetists. In contrast the incidence of breast cancer in England and Wales among women of all ages is 65.9/100 000²⁴—that is, there was almost a 50-fold increased incidence. An excess of cancer among male anaesthetists has also been found in a prospective study,²⁵ although the statistical interpretation of these data has often been disputed despite the results being not significantly different from those of a retrospective study by the same workers.²⁶

Statistical critics⁵⁻⁷ insist on a level of proof beyond all reasonable doubt; a probability of 0.05 is often cited, yet when this appears it is either ignored⁶ or dismissed as a quirk.²⁷ This is the level of proof required when, for example, benefit is claimed from a new treatment. The legal analogy is where the risk of a false conviction is particularly abhorrent and so the standard of proof required is beyond reasonable doubt. Just as abhorrent is a spurious claim of safety and the risk of a type II error; accepting a false claim must therefore be minimised. The legal analogy is a false acquittal. Use of the null hypothesis⁵⁻⁷ tends to maximise the risk of a type II error. To insist on a level of proof beyond all reasonable doubt before taking preventive measures is to prejudice those at risk to no advantage and could be considered irresponsible. Failure to recognise the consequences of a type II error²⁸ is a feature of several critiques.⁵⁻⁷ Other workers,^{14 18} also using the null hypothesis, have presented their conclusions simply as "not significant" and not given a probability value. Notwithstanding this, my results were beyond all reasonable doubt.

The results suggest that chronic exposure to pollutant anaesthetics may induce mutations affecting principally the musculoskeletal or central nervous system or that it interferes with the protective codons (units in the DNA genetic code) during meiosis, allowing the emergence of recessive anomalies from within the genetic pool and hence the emergence of unusual but well-known disorders that have a familial background. Some anaesthetics interfere with DNA in plants²⁹ and man to such an extent that the changes may be determined biochemically³⁰ even after comparatively brief exposure. Since any mutagenic agent is likely to be carcinogenic it is not surprising that evidence of carcinogenicity appeared. Several anaesthetics are carcinogenic.³¹

Part of my study was to assess ventilation in places where anaesthetics are given. Where ventilation equipment exists it seems reasonable to institute regular checks on its performance. The fact that ventilation systems are often inadequate or non-existent suggests that active scavenging systems should be installed as a stopgap measure. These systems at best are inefficient means of removing all pollution,³² especially if the theatre is heavily contaminated, but since the risk to health is apparently so great any gas removed is likely to be beneficial. Scavenging systems deal only with gas emitted from the expiratory valve, but there are many sources of pollution arising from anaesthetics—for example, no anaesthetic circuit in regular use remains totally gas tight; furthermore, all patients immediately after any anaesthetic of modest duration exhale

20-30 litres of nitrous oxide into the operating theatre within the first five minutes.

Current ventilation recommendations in operating theatres of 18-20 air changes an hour are based on bacteriological and heat balance considerations. There is no evidence that these changes per hour are effective at abolishing pollution. Dead pockets of contaminated air may persist despite the ventilation equipment performing to specification. Unless careful attention is paid to air-flow patterns produced by the ventilation equipment some of the fresh air will blow straight through as a draughty beam of air to the exit ports, and hence the room will not be properly scoured of pollutant gas.

Apart from scavenging devices other measures to reduce pollution include a greater use of local anaesthetics. Up to 30% of routine operations may be performed under local anaesthesia,³³ but for this to gain acceptance substantial public and professional education will be needed. Purely intravenous means of maintaining anaesthesia are still in their infancy. Closed-circuit anaesthetic systems, however, reduce pollution only from the expiratory valve and are therefore of little help when scavenging systems are available.

Despite the suggestion that women anaesthetists should stop work if pregnant,³⁴ my results suggest that male anaesthetists are almost equally vulnerable. There are economic and social pressures for young anaesthetists to remain at work—even if they are trying to beget a family—as there is no alternative paid employment for them. Sympathetic rearrangements of their duties by administrative anaesthetists could be of some help. The best solution appears to be good ventilation as used in orthopaedic surgery, and the useful offshoot would be reduced wound infection.

Finally, the observations of affected children among the older anaesthetists suggest that the problem has been with us for a long time and that it is only the recent explosive growth in the number of anaesthetists that has brought it into focus. Equally the only inhalational anaesthetic common to the whole survey period was nitrous oxide, although all anaesthetics will induce abnormalities if enough is given.

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Occult aortic stenosis as cause of intractable heart failure

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Summary and conclusions

During a three-year period 10 patients with critical aortic stenosis were referred to a cardiac referral centre with symptoms and signs of intractable cardiac failure and low cardiac output. In nine patients the correct diagnosis was not suspected at the referring hospital, and in the remaining patient the true severity of the aortic stenosis was not appreciated and cardiomyopathy was suggested as an additional diagnosis. The most common referral diagnoses were severe mitral regurgitation (four patients), congestive cardiomyopathy (two patients), or both (three patients). Only two patients had soft ejection systolic murmurs at the base of the heart radiating into the neck, and such a murmur appeared in a third patient during medical treatment. The carotid pulses were of small volume but the characteristic slow-rising, anacrotic nature of the pulse could not be appreciated clinically. The diagnosis was suspected in nine patients because of aortic valve calcification detected by lateral chest x-ray examination in seven patients and by x-ray screening of the heart in two, and because of abnormal aortic valve echoes in the echocardiogram of all five patients in whom the aortic valve could be seen. Eight patients underwent aortic valve replacement despite seemingly poor pre-operative left ventricular function. Three patients died, of whom two had severe coexistent coronary artery disease. The five survivors all returned to normal lives and needed little or no medication.

Critical aortic stenosis should be actively sought in patients with severe heart failure of unknown cause since surgery may enable them to resume their normal lives.

Introduction

Aortic valve stenosis is usually easily recognised on the basis of well-established and characteristic symptoms and physical signs, and surgical treatment now achieves satisfactory results with low mortality.¹⁻³ Recognising aortic stenosis clinically and assessing its severity may become difficult if left ventricular

dysfunction supervenes. As cardiac output falls and the flow across the stenosed valve decreases the characteristic murmur becomes softer or disappears, the slow-rising pulse is more difficult to appreciate, and the clinical picture becomes that of low cardiac output with pulmonary oedema and heart failure.

We report on a series of 10 patients who presented with severe heart failure refractory to medical treatment and in whom the correct diagnosis of critical aortic stenosis was not suspected at the referring hospital. We present these cases to emphasise the features that led to the correct diagnosis being suspected.

Patients, methods, and results

CLINICAL PRESENTATION

Table I shows the clinical features of the 10 patients studied, who presented at this hospital between 1974 and 1977. In only one patient (case 6) was the true diagnosis suspected at the referring hospital, and even then a superadded cardiomyopathy was suggested as the signs of obstruction of the left ventricular outflow tract were not impressive. The remaining patients were referred as cardiac failure of unknown cause. Mitral regurgitation alone (four patients) or in association with cardiomyopathy (three patients) was the most commonly suspected underlying cause of the cardiac failure. This was because these patients had apical systolic murmurs, which in several cases may have been due to functional mitral regurgitation since in four this was subsequently shown at cardiac catheterisation. In two patients congestive cardiomyopathy alone was suspected. All patients had severe (New York Heart Association (NYHA) grade IV) symptoms, evidence of reduced cardiac output with a small carotid pulse, and a cool periphery despite intensive medical treatment. Three patients had a soft ejection systolic murmur, best heard at the base and radiating into the neck; in one of these patients this appeared during medical treatment.

INVESTIGATIONS

ECG (table I)—The ECG in five patients showed increased QRS voltage, depressed ST segments, and lateral T-wave inversion, which suggested the possibility of aortic stenosis. The changes in the other patients were non-specific, although in three cases there was impaired conduction (left bundle-branch block, left bundle-branch block with first-degree heart block, and left axis deviation with first-degree heart block).

Chest x-ray examination—Posteroanterior chest x-ray films showed cardiomegaly and pulmonary oedema in all patients. Lateral chest x-ray films suggested aortic valve calcification in seven patients (table I), which was confirmed by screening using a high-resolution caesium-iodide image intensifier (CGR). Screening also showed aortic valve calcification in two patients in whom it was not detected on the lateral chest x-ray film. The one patient in whom aortic valve calcification was not detected also had non-specific ECG changes (case 4).

Echocardiography—An M-mode echocardiogram was obtained in six patients. This showed an enlarged left ventricular cavity with poor

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