cost of £2 per smear might be reasonable at the present time. Thus a sum of £6000 has been spent, the cytological services have examined 3000 normal smears from an age group where the chances of positive findings are notably low, and little has been achieved.

The difficulty is that publicity in women's magazines together with the policy of the Family Planning Association and the Brook Clinics make those who do not do routine smears in young people appear negligent, and it is as quick to take a smear as to explain one's reasons for not doing so. There is a need now for publicity to be given in non-medical magazines to Drs Spriggs and Husain's paper.

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SIR,—I write further to the communication from Dr A I Spriggs and myself (11 June, p 1516) relating to the age and frequency of screening for cervical cancer in order to bring out a further point of some significance.

The National Screening Programme, now in its 13th year with an annual load of around 3 million smears, was originally designed to test women aged over 35 once every five years. As a result of the open-door policy, the enthusiasm of the young, and the use of maternity and family planning clinics in which to collect the smears 50% of the tests entering our laboratories are from women under the age of 35. This has resulted in the application of about 1.5 million tests to about 6 500 000 young women, whereas the other 1.5 million tests have been spread over nearly 13 000 000 women with a diminishing frequency as they get older. (Figures taken from *Registrar General's Statistical Review of England and Wales, 1973, Part II*.)

It would be expected that a substantial number of women at risk would need to be

screened for a substantial part of the precursor phase before one could expect the mortality rate of the subsequent cancer to decline. The authors of the Walton Report¹ have shown this to be so in British Columbia, where about 80% of the population has now been screened, which probably relates to about 60% of the total group at risk as these seem to accumulate in the unscreened population.

Looking at the age-specific mortality rates of cervical cancer quoted by J G Thomson in his article in *Health Trends* in 1971² one could see that there was a considerable drop in mortality in women in the 35-44-year age group but in no other. In my lecture to the Marie Curie Foundation in 1975 (in press as a triennial report) I had suggested that this could have been due in part to the screening programme. The more recent figures provided by kind permission of Dr A M Adelstein of the Office of Population Censuses and Surveys (table I) demonstrate this effect even more clearly, with the drop in mortality extending into the 45-54-year age group.

The other explanation was, of course, that a lower-risk cohort had emerged over the past few years or that the disease was losing its aggressiveness. Table II does indicate that this drop could be due to cohort differences, but may not explain the whole effect (data kindly supplied by Dr Adelstein). Indeed, there appears to be a younger cohort following up behind in which the rate is rising again, and it is for this reason that, in our recent article, we suggest in our model proposition for a screening schedule that we should take note of any mortality and morbidity trends, especially in view of the changing life style of the young women of today.

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¹ Walton Report, *Canadian Medical Association Journal*, 1976, **114**, 1003.

² Thompson, J G, *Health Trends*, 1971, **3**, 24.

TABLE I—Deaths from cancer of the cervix uteri

Year	Age group (years)						All ages
	0-24	25-34	35-44	45-54	55-64	65 and over	
1963	1	31	316	561	570	986	2465
1964	3	32	337	627	561	1017	2577
1965	3	33	330	596	604	887	2453
1966	2	27	280	535	599	1021	2464
1967	3	32	241	628	574	971	2449
1968	3	32	226	630	618	925	2434
1969	6	32	205	564	607	1003	2417
1970	5	34	170	566	625	1003	2343
1971	2	41	184	580	594	914	2315
1972	4	46	164	510	604	890	2218
1973	7	39	137	542	605	919	2249
1974	5	50	122	443	524	924	2068
1975	9	68	136	412	597	921	2143

TABLE II—Cohort mortality from carcinoma of the cervix in England and Wales

Central date of birth	Death rate (per million) in women aged									
	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74
1881										315
1886										313
1891										274
1896										247
1901						203	254	285	284	274
1906					136	181	197	246	232	247
1911					154	187	178	193	193	206
1916				93	180	204	199	199		
1921			58	134	176	190				
1926		30	74	106	130					
1931	10	37	67	67						
1936	9	18	44							
1941	5	15	38							
1946	7	22								
	10									

Anaesthetic waste gas scavenging systems

SIR,—I was very interested to see the letter from Professor L Rendell-Baker and Dr R A Milliken (21 May, p 1348) on the above subject. They came to the same conclusion as we did (19 February, p 507), that an effective scavenging system used without adequate ventilation will not correct pollution problems in operating theatres. We have suggested two additional measures which are necessary to achieve effective control of ambient pollution—that is, regular equipment maintenance with careful anaesthetic techniques to prevent gas leaks and institution of an air monitoring programme to indicate the effectiveness of these preventive measures. Regular equipment maintenance involves frequent testing of pipe lines, anaesthetic machines, ventilators, and scavenging systems for gas-tightness.

However, I am surprised by their statement that passive systems are difficult to use since the driving power, the positive force of expiration, and circuit overflow also tend to propel the pollutants into the theatres. This has not been our experience. In our study,¹ we were able, with the help of a passive scavenging system and operating room ventilation with an air exchange rate of 10 times per hour, to reduce halothane levels to the order of 0.2 ppm. This was achieved irrespective of the type of anaesthetic circuit used and gas flows and vapour concentration employed. In our recent study² on passive disposal of expired anaesthetic gases the results showed that the resistance offered by the conduction channels and the terminal gas exhaust valve of the passive disposal system was extremely low. It was found to be 25 Pa at a gas flow rate of 30 l/min. Provided the various components and joints of the passive scavenging system are gas-tight there is no reason why it should be less effective than any active system in keeping operating rooms pollution-free.

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¹ Mehta, S, et al, *Canadian Anaesthetists' Society Journal*, 1975, **22**, 271.

² Mehta, S, et al, *British Journal of Anaesthesia*, 1977, **49**, 589.

Effect of antihypertensive drugs on growth hormone secretion

SIR,—The report by Drs I Lancranjan and P Marbach (4 June, p 1472) on the failure of BS 100-141, a new α -adrenoceptor agonist, to raise plasma growth hormone (GH) levels in hypertensive patients after both acute and chronic treatment prompts us to report our experience with clonidine, another central α -adrenoceptor agonist which was found to increase GH secretion in young normal male volunteers.¹

Seven patients aged 40-60 years with moderate hypertension volunteered for the study. There were five men and two women. All patients had discontinued their antihypertensive treatment for at least one week and took no medication other than clonidine during the study. On the first day clonidine (0.15 mg diluted in normal saline) was infused intravenously over a 10-min period and blood was drawn for GH radioimmunoassay at 15-min intervals for 90 min. On the following day 0.15 mg of clonidine was administered orally and blood was taken at hourly intervals for 3 h. This test was followed by treatment with clonidine