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Influenza vaccination and the elderly

Offer it to elderly people in whom longevity is a blessing

Each autumn vaccine manufacturers and the media focus our attention on influenza vaccines, and recommendations on their use are issued annually by the chief medical officer. Immunisation is not recommended for the attempted control of the spread of influenza; rather it should be considered for groups thought to be at special risk, including elderly patients who have chronic pulmonary, cardiac, or renal disease; diabetes; or other endocrine disorders and conditions treated with immunosuppressive agents and those living in residential homes and long stay hospitals.[1] But how serious a threat is influenza? How effective are the vaccines, and are they safe?

The incidence of deaths from cerebrovascular, cardiovascular, and respiratory diseases increases during the winter, and this seasonal variation is influenced by low temperature and respiratory viruses such as influenza and respiratory syncytial virus.[2-6] Additional deaths above the normal winter increase are recorded regularly in association with influenza epidemics: 10 000 or more excess deaths were documented in each of 19 epidemics in the United States from 1957 to 1986[7]: about 120 000 excess deaths were attributed to influenza in England and Wales during the 10 winters after influenza A/Hong Kong (H_3N_2) first arrived[8]; none were recognised in the United Kingdom during seven consecutive winters from 1978-9 to 1984-5,[9] but 26 080 were identified in England and Wales last winter, although only 2440 were certified as being due to influenza and a further 5260 to pneumonia.[10] Generally about half the excess deaths during influenza epidemics are attributed to influenza, bronchitis, and pneumonia and many of the remainder to cerebrovascular and cardiovascular disease—implying that influenza is responsible for many hidden deaths.

The risk factors for fatal influenza are age and underlying disease. About 80-90% of the excess deaths are among people aged 65 or over. In England and Wales during the 15 years 1974-88 the lowest mean mortality for deaths certified as due to influenza (0·04/100 000) occurred in 5-14 year olds; mortality increased in successive 10 year age bands by threefold, fourfold, sixfold, 11-fold, 32·5-fold, 100-fold, and 765-fold to a mean of 30·6/100 000 (range 3·4-190·2) in those aged 75 or over (World Health Statistics Annuals 1977-89). In people aged 45 or more the presence of chronic medical disease increases death rates from pneumonia and influenza by at least 39-fold; cardiovascular, pulmonary, and combined cardiovascular and pulmonary disease increases the risk by 104-fold, 240-fold, and 870-fold respectively.[11] The toll of upper respiratory tract infections in old people's homes may be substantial: in Leicester during 1988-9, a non-epidemic year, about one in 30 residents of these homes with symptomatic colds died.

The composition of influenza vaccine is changed almost every year so that it contains the strains most likely to be effective. Whereas the vaccine can offer 60-80% protection to normal healthy adults when vaccine and epidemic strains are closely related, a review of 16 studies in geriatric homes since 1972 showed a mean protection against influenza-like illness of only 27% for influenza A (H_3N_2) vaccines. Influenza B vaccines fared even worse, with a mean protection of only 21% in seven studies.[12] Moreover, Feery *et al* found no protection against virologically proved cases of influenza A/Victoria/3/75 in elderly people in residential homes in Australia.[13] Of greater relevance, however, are the considerable reductions in the incidence of bronchopneumonia (49-90%; mean 69%), admissions to hospital (47-72%; mean 59%), and deaths (0-100%; mean 69%) among elderly subjects who have been vaccinated during influenza outbreaks when the vaccine and epidemic strains were closely related.[12-18]

Surveys of elderly people, both those in residential homes and those who live at home, suggest that almost two thirds have one or more chronic medical conditions (Market and Opinion Research Institute poll conducted for the Influenza Monitoring and Information Bureau, April 1990),[6] yet despite the evident benefits of influenza vaccine in the elderly, vaccine is given to less than one fifth of the total elderly population in Trent,[19] less than a half of the residents of old people's homes in Leicester,[6] and to patients in continuing care wards in Britain by fewer than one consultant geriatrician in five.[20] The reasons for poor vaccine distribution include scepticism about its efficacy, concern over its safety, expense, and the view that it is inappropriate or unnecessary. Until the late 1960s local and systemic adverse reactions to influenza vaccines were common and at times severe, but with the introduction of new purification techniques few recipients now have local or generalised reactions. Detailed studies of the cost efficacy of influenza vaccine in elderly populations have not been carried out, but extrapolation of data from Leicester and Glasgow suggests that vaccination of all residents in long stay homes would not be economically worth while unless there were regular epidemics.[6 20] Even then the calculations ignore the dilemma of vaccinating patients in whom the quality of life is especially poor and the actual cost of maintaining patients in homes or wards. On the evidence, then, doctors should certainly offer vaccine to elderly people in whom longevity is a

blessing. For the remainder, doctors should consider the herd immunity that evidently accrues in homes with high immunisation rates[21] and then wrestle with their consciences.

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Vasectomy and the human testis

We still know too little about the effects of vasectomy

Concerns about vasectomy have so far focused on its reversibility[1-3] and fears that it might predispose to cardiovascular disease.[4,5] But a recent study by Cale *et al* has raised a new and serious worry: that vasectomy might accelerate the growth of testicular tumours.[6]

Of a cohort of over 3000 men in central Scotland who had undergone vasectomy, eight developed testicular cancer within four years after the operation—compared with an expected 1·9.[6] As the authors did not indicate the types of tumours we do not know whether this finding was just an unhappy coincidence (as different tumour types would suggest) or whether we should be more worried (as similar pathological appearances would suggest).

This is not the first suspicion of accelerated testicular tumour growth after vasectomy. Thornhill *et al* in Dublin reported three cases of a comparatively rare mixed seminoma and malignant teratoma within eight weeks after surgery.[7] Strader *et al* reported an increased incidence of testicular cancer among Catholic but not among non-Catholic men in Washington state,[8] but they attributed the difference to a failure to report vasectomy by Catholic controls in the questionnaire study.

These observations may be chance findings by rightly cautious practitioners, and there is insufficient evidence to implicate vasectomy in accelerated tumour growth. If vasectomy does promote such growth it is not clear how it does so. Indeed, its general effects on the human testis are controversial and incompletely understood.

Animal studies have made it clear that there are considerable differences in the effects of vasectomy among species. Dogs show temporary depression of spermatogenesis, which may be related to raised intraluminal pressure[9-12]; guinea pigs suffer autoimmune orchitis with infiltration by leukocytes[13]; rabbits develop degeneration of the seminiferous epithelium associated with deposition of immune complexes along the basement membrane[14,15]; and rats, rabbits, and hamsters all show testicular atrophy associated with the formation of sperm granulomas in the caput epididymidis.[16-18] Which, if any, of these models applies to man is not known.

Several groups have reported finding abnormalities in testicular biopsy specimens in some men after vasectomy.[19-25] The changes are variable but include degeneration of seminiferous epithelium; loss of germ cells, especially spermatids; dilatation of testicular tubules; thickening of tubular walls; and interstitial fibrosis. The causes are not known. Dilatation of seminiferous tubules suggests raised intraluminal pressure,[24,25] which might account for the epithelial changes. Raised intraluminal pressure has been detected in the seminiferous tubules of guinea pigs given vasectomies.[26] Rats with vasectomies, although usually showing normal testes,[27] develop appreciable distension of seminiferous tubules if the caput epididymidis becomes obstructed.[17,18]

Some of these changes may be reversible. Three groups of workers have shown that some men who undergo reversal of vasectomy are subsequently fertile despite showing pronounced degeneration on testicular biopsy at the time of reversal.[22,24,25] In dogs the depression of spermatogenesis after vasectomy is only temporary and may be attributable to raised intraluminal pressure.[9-12] Perhaps the seminiferous epithelium of the dog adapts more readily to raised pressure than that of man and regressive changes in humans are reversed only when the ductus deferens is reanastomosed. The presence of interstitial fibrosis in biopsy specimens taken at the reversal of vasectomy may carry a poor prognosis for fertility.[24]

Bigazzi and Alexander and Tung, working on rabbits with vasectomies, reported degeneration of seminiferous epithelium associated with immune complex deposition in the basement membrane; elution techniques showed that the complexes contained antisperm antibodies.[14,15] Because just under two thirds of men who have undergone vasectomy develop serum antisperm antibodies[28,29] it has been suggested that the thickening of the tubular walls seen in testicular biopsy specimens from such patients may represent the same process, but Bigazzi *et al* could not show any immune complex deposition.[23]