High frequency oscillation and tracheobronchial clearance

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The paper of van Hengstum c.s. describes the inefficacy of oral high frequency oscillation (HF) combined with forced expiration manoeuvres on tracheobronchial clearance in patients with chronic bronchitis (1). In our experience with a slightly different technique, using an endotracheal catheter, oscillating frequency of 1.66-2.50 Hz and a stroke volume of 70-100 ml, a 3 to 5 fold increase in sputum production was found (2). The difference in results may be due to technical differences but also to a difference in patient characteristics. A major disadvantage of the technique of van Hengstum c.s. may be the oral administration of oscillating waves, because the force of these waves may be dampened by the flaccidity of the oropharyngeal cavity and the narrow laryngeal entrance. The weekly introduction of an endotracheal catheter with topical anaesthesia was well tolerated by our patient. Our patient with bronchiectasis had a normal specific compliance; the compliance of the 8 bronchitics of van Hengstum c.s. is not reported. In our experience (unpublished data) the results of HF in patients with a flaccid lung are disappointing. This is explained by the following theory. The efficacy of HF on the clearance of the bronchial tree depends on the capacity to mobilize sputum and on the capacity to produce a sufficient expelling gas flow. The cleansing model of an effective cough comprises two, main factors:

1. the preparation of the sputum for transportation by disrupting its adherence to the bronchial wall. Three mechanisms are involved: a. a tearing force due to the driving power of the gasflow; b. a whirling force due to the turbulence of the gas in the larger airways; c. a vibrating force due to the oscillations of the system causing standing oscillations of the gas which have a tearing effect and vibrations of the bronchial wall loosening adherent sputum. 2. the transport process in which the magnitude and the velocity of the gas flow determine the efficacy of the expelling force. Using a high resolution pneumotachograph Douma showed the presence of high frequency oscillations during coughing, perpendicular on the standing oscillations of the gasflow (3). These oscillations were attributable to the stiff elements of the system i.e. the bronchial wall. They were absent or greatly dampened in patients with flaccid lungs. It is clear that in patients with flaccid lungs two main factors promoting effective clearance of the bronchial tree are lacking: 1. the gas flow is markedly reduced and therefore its tearing and expelling force; 2. the flaccidity of the structure causes a dampening of the vibrations, interferring with the loosening of the sputum. Both conditions are not improved by high frequency oscillation treatment. We think that the results of van Hengstum c.s. can not be judged properly without knowing the pulmonary compliance of their patients. In patients with emphysema HF may even be contraindicated because the jetflow may over distend their flaccid lungs (4).

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Alveolar macrophages from smokers show strong intracellular fluorescence

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With great interest we read the well documented case report by Dr Parra et al (1). The authors described the development of interstitial pneumonitis due to amiodarone treatment in a male smoker. The drug administration was discontinued and treatment with oral steroids caused a dramatic improvement. However, two months after cessation of steroid therapy a relapse occurred. Open lung biopsies showed a granular autofluorescent material in the cytoplasm of interstitial and alveolar macrophages (AMs) when examined under ultraviolet (W) microscope.

However, it has been known for some years that alveolar macrophages (AMs) are fluorescent (2) and we

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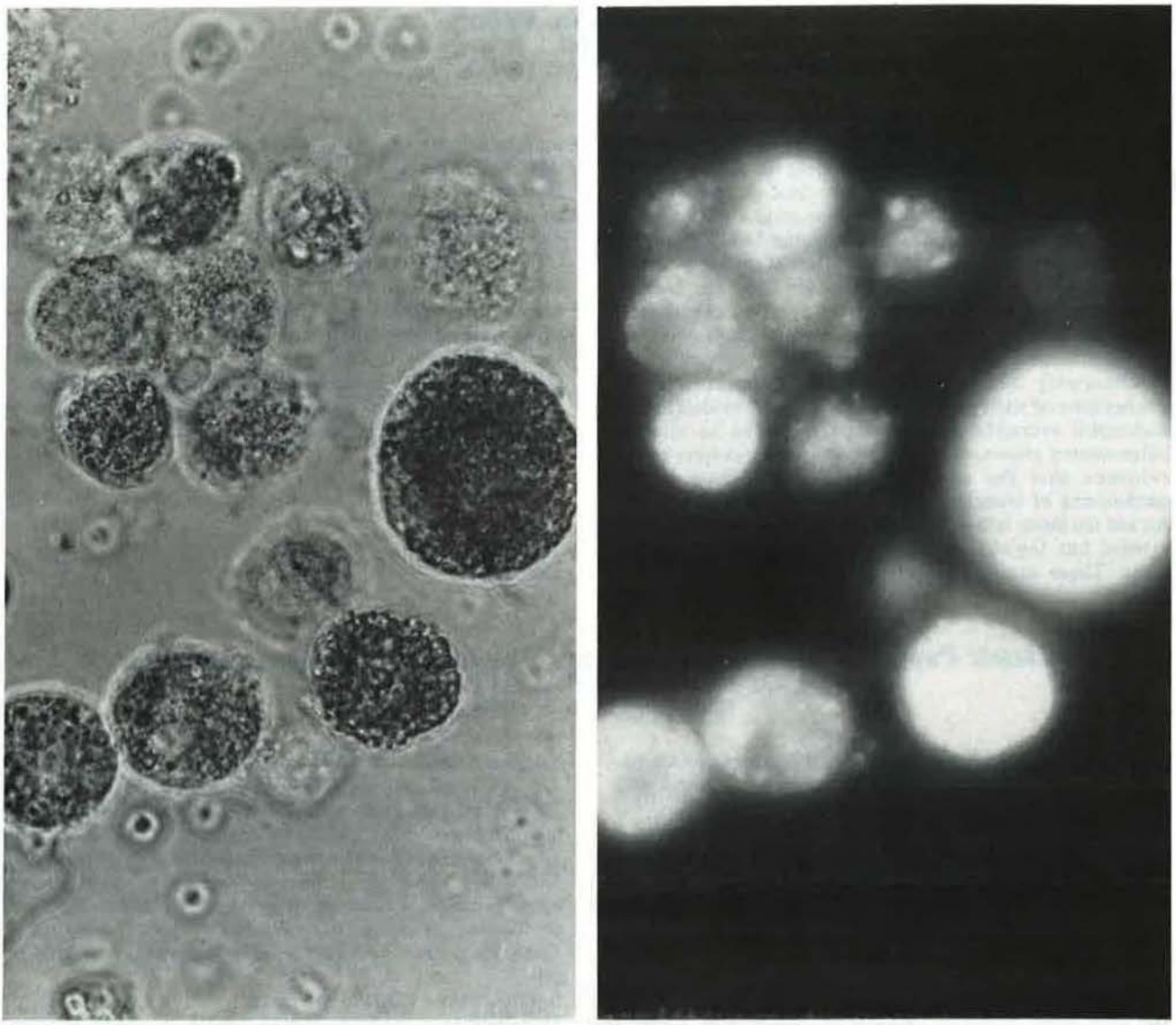

Fig. 1. - Alveolar macrophages from a smoker in a light microscope (left) and the corresponding cells in a fluorescence microscope (right).

have recently shown that AMs from smokers show a fourfould increased mean fluorescence intensity compared to AMs from non-smokers as measured by flow cytofluorometry (3). In a UV -microscope this autofluorescence has a granular pattern as shown in Fig 1. The strong fluorescence in AMs from smokers is above all due to phagocyted fluorescent material from tobacco smoke (unpublished observation).

The patient described by Dr Parra et al was a smoker (12 packyears) and one possible explanation for the finding of autofluorescent material in his AMs could be his smoking habits. Even if the patient had stopped smoking at the time when he became ill, the fluorescent material can be found a long time after smoking cessation. In an ongoing study (4) we found that autofluorescence remained high in AMs from smokers more than 9 months after cessation of smoking.

In conclusion, we believe that the patients smoking history should also be considered when the possible mechanisms behind the increased autofluorescence in the AMs are discussed.

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