# THE DEPOSITION OF FIBERS AND SPHERES AT THE CARINA IN EXCISED LUNGS

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## INTRODUCTION

The deposition of dusts in a respiratory system is a function of the airflow characteristics and the aerodynamic behavior of the particles within the system. The deposition of spheres in the human respiratory track has been studied both empirically and theoretically.[1] Thus, several models exists for the deposition of compact particles in regions of the respiratory system. However, the information on the deposition of fibers is relatively lacking. The correspondence of the mathematical models available to predict the deposition of fibers in the human respiratory tract to the deposition models for compact particles suffer due to the limited empirical knowledge of fiber deposition available. Studying the comparative deposition of fibers and spheres at the carina can provide valuable insight into the deposition of fibers in the tracheo bronchial system. The aim of this study was to investigate the comparative deposition of fibers and spheres at the carina experimentally.

## EXPERIMENTAL METHODS

The basic principle of the experimental method is the assimilation of the natural breathing in an excised calf lung. To accomplish this end, the excised lungs were caused to inspire and expire by varying the pressure around them by placing them into a variable pressure apparatus consisting of a sealed chamber connected to a respiration pump. The pump controlled both the breathing rate and the tidal volume by systematically withdrawing and replacing air from the chamber housing. On inspiration the resultant decrease in pressure around the lung caused it to expand until the intra alveolar pressure equilibrated with the new pressure in the chamber. The pumps full cycle was complete when the withdrawn air was replaced returning the chamber to its original atmospheric pressure. Every experiment utilized 15 respirations per minute. Typically, the tidal volumes generated were between 400–500 cc.

Nearly monodisperse, size classified glass spheres and glass fibers prepared by using the method described by Esmen et al.[2] were used as the deposition material. The dust generator used in this experiment consisted of a dust reservoir, a clapper, and tubular delivery system. Before reaching the trachea air the stream was split in two by a copper bifurcation to provide for a sampling port. The sampling port was used to measure the airborne concentration of the particles during each experiment. This sampling rate was equal to the lung's tidal volume and was drawn simultaneously with lung inspiration.

The 29 pneumonia free calf lungs used in this experiment were obtained at the time of slaughter. After carefully excising the lung carcass, the surrounding tissues and organs were removed. The lung surface was rinsed and inspected for cuts and rips and the lungs were kept moist until the end of the experiment. In the final preparation, the trachea was cut about 18 cm above the carina and two ring clamps were placed on the trachea just above the right apical bronchi. An artificial tracheal extension was inserted into the trachea and secured by the ring clamps. The entire preparation was seated inside the variable pressure unit with the artificial trachea passing through a hole in the top of the chamber connected a leg of copper bifurcation. The lung, inspiring and expiring with the changes in chamber pressure was connected to a spirometer. The tidal volume was monitored for 3–5 minutes. The tidal volume usually stabilized within 1–2 minutes. The dust generator was started and synchronized such that a clap on the dust reservoir occurred simultaneously with the onset of inspiration. The exposure was about 20 minutes.

The experimental section was separated from the rest of the lung by carefully cutting away the surrounding parenchymal tissues and then cutting the bronchi about one inch distal to the carina. This portion was carefully cleaned of adhering fat and parenchymal tissue and frozen. In order to minimize particle translocation, all subsequent cutting was performed on the frozen tissue. The trachea was sliced into two sections for analysis of deposited particles. The first slice was made just under the right apical bronchi. The second section which included the carina was taken after slicing 1.5 cm posterior to the carinal plane. The removal of the particles from the tissue was achieved by sonication and subsequent ashing. The ashed material was redeposited on a filter for analysis. The filters were viewed under cross polarization. If the total number of spheres or fibers deposited on the filter was less than about 2000, then the entire filter was viewed and all particles counted. Generally, an analysis of 1 $cm^2$ was sufficient. Some of the particles were lost during the transfer and processing operations. A calibration was performed to delineate the lost fraction.

## RESULTS AND DISCUSSION

A list of the experimental parameters are presented in Table

Table I

The Experimental Conditions and Parameters

| Lung | Size | | | Tidal Volume | Tracheal Velocity | Stokes' Number(*) | |
| | Sphere Dia. | Fiber Dia. | Length | | | | |
| | um | um | um | liter | cm/sec | Sphere | Fiber |
| --- | --- | --- | --- | --- | --- | --- | --- |
| 12 | 24.5 | 10.9 | 50 | 0.330 | 34 | 0.126 | 0.054 |
| 13 | 24.5 | 10.9 | 50 | 0.355 | 36 | 0.133 | 0.057 |
| 15 | 24.5 | 10.9 | 50 | 0.355 | 55 | 0.232 | 0.104 |
| 16 | 24.5 | 10.9 | 50 | 0.330 | 77 | 0.396 | 0.185 |
| 17 | 24.5 | 10.9 | 50 | 0.380 | 55 | 0.243 | 0.104 |
| 18 | 24.5 | 10.9 | 50 | 0.430 | 68 | 0.315 | 0.134 |
| 19 | 24.5 | 10.9 | 50 | 0.330 | 93 | 0.574 | 0.245 |
| 21 | 24.5 | 10.9 | 50 | 0.380 | 45 | 0.181 | 0.082 |
| 22 | 24.5 | 10.9 | 50 | 0.430 | 72 | 0.333 | 0.146 |
| 30 | 12.5 | 6.4 | 48 | 0.400 | 58 | 0.063 | 0.051 |
| 32 | 12.5 | 6.4 | 48 | 0.475 | 134 | 0.202 | 0.164 |
| 34 | 12.5 | 6.4 | 48 | 0.550 | 103 | 0.124 | 0.101 |
| 36 | 12.5 | 6.4 | 48 | 0.575 | 87 | 0.095 | 0.078 |
| 38 | 12.5 | 6.4 | 48 | 0.500 | 98 | 0.124 | 0.101 |
| 39 | 12.5 | 6.4 | 48 | 0.525 | 111 | 0.141 | 0.115 |
| 40 | 12.5 | 6.4 | 48 | 0.475 | 65 | 0.068 | 0.055 |
| 41 | 17.2 | 9.1 | 50 | 0.505 | 85 | 0.199 | 0.143 |
| 42 | 17.2 | 9.1 | 50 | 0.565 | 111 | 0.282 | 0.202 |
| 43 | 17.2 | 9.1 | 50 | 0.485 | 58 | 0.110 | 0.079 |
| 44 | 17.2 | 9.1 | 50 | 0.500 | 69 | 0.143 | 0.103 |
| 45 | 17.2 | 9.1 | 50 | 0.505 | 82 | 0.187 | 0.134 |
| 46 | 17.2 | 9.1 | 50 | 0.525 | 70 | 0.128 | 0.092 |
| 47 | 17.2 | 9.1 | 50 | 0.555 | 88 | 0.183 | 0.131 |
| 48 | 17.2 | 9.1 | 50 | 0.570 | 101 | 0.243 | 0.174 |
| 49 | 17.2 | 9.1 | 50 | 0.525 | 81 | 0.176 | 0.126 |
| 50 | 17.2 | 9.1 | 50 | 0.525 | 92 | 0.210 | 0.151 |
| 51 | 17.2 | 9.1 | 50 | 0.515 | 86 | 0.196 | 0.141 |
| 55 | 12.5 | 6.4 | 48 | 0.560 | 123 | 0.165 | 0.134 |
| 56 | 12.5 | 6.4 | 48 | 0.575 | 96 | 0.119 | 0.097 |

(*) The Stokes' numbers for the fiber diameters Df and aspect ratio B is calculated by the use of impactive diameter Di using the formula (3):

$$Di = Df (1+0.013(\ln B)^3 (0.71 + 0.91 \ln B)^{1/2}$$

calculated using impaction diameter formulation developed by Burke and Esmen.[3] The graphical representation of the deposition efficiency as a function of tracheal velocity for the lowest group of Stokes' number particles is shown in Figure 1. These results indicate that a consistent deposition occurs at low velocities; seemingly independent of the velocity. This may be explained by theory developed by Harris,[4] who predicted such an effect would occur due to interception. As the tracheal velocity increases, the slope of the curve changes rapidly, indicating that a critical point was reached permitting the impaction to facilitate deposition rapidly.
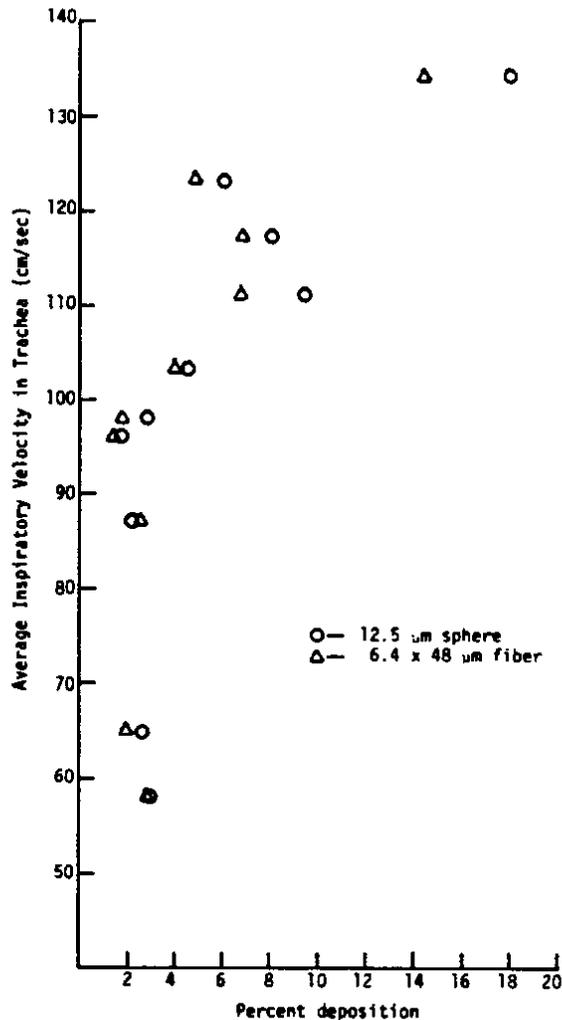


Figure 1. Deposition of fibers and spheres as influenced by inspiratory velocity.

The critical point was investigated by incorporating the air flow characteristics with particle physical parameters and observing the depositional efficiencies as a function of Stokes number. Such a graph of all results is provided in Figure 2. In this figure, data of impactive deposition as observed by Johnston and Muir[5] and Landahl and Herrmann[6] are also included.
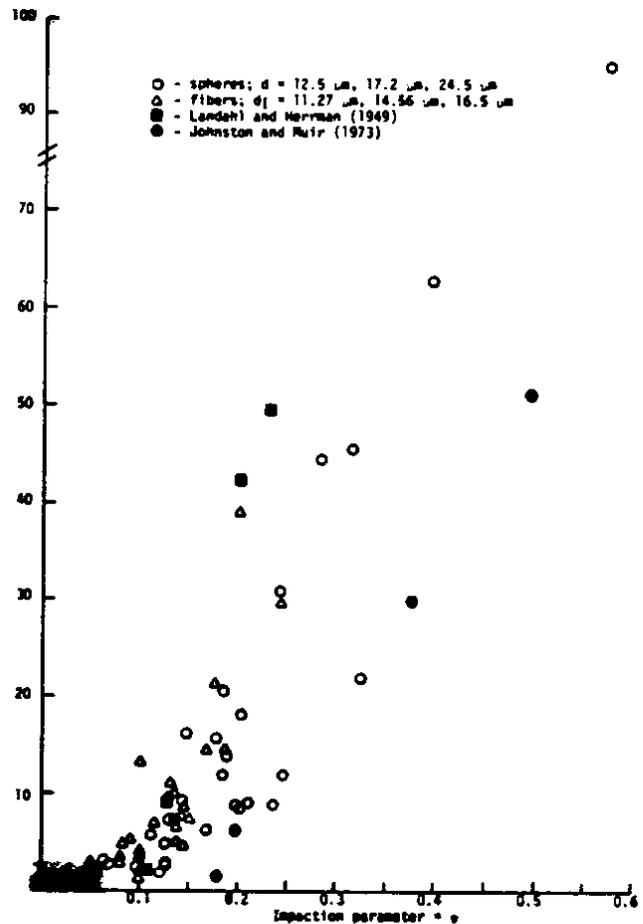


Figure 2. Deposition of fibers and spheres as a function of Stokes' number.

Clearly, during the experiments, impaction was occurring simultaneously with particle removal by other mechanisms. By the use of current theories, estimates of depositional efficiency due to the other mechanisms may be made. Harris noted that interception is an effective removal mechanism in the tracheobronchial region for fibers as small as 10 μm in length. If interception were significant in these experiments then one would expect a uniform shift to the left on the deposition curve (Figures 1 and 2); because, the amount removed would be independent of all parameters except airway and particle size. The results shown in Figure 1 suggests that the interception would account for 2–4 percent deposition.

Similarly, if sedimentation were a significant mode of particle removal the shift would not be uniform. Rather it would be biased in favor of those experiments involving large particle sizes and low average tracheal velocities. Review of the data does not provide any evidence of such bias. Harris' equation for settling in turbulent flow suggests that only 2 percent of the largest fibers which experienced the lowest average tracheal velocity would settle at the carina.[4]

If the impaction efficiency for round jets impinging upon an impaction surface perpendicular to the flow axis is taken to be 0.5 for Stokes' number about 0.25,[7] then the results suggest that the impaction efficiency observed is significantly less than what would be expected. In fact a recent theoretical work on impactive deposition of fibers this deviation was also noted.[8] There is strong evidence indicating that interception, and sedimentation may be augmented by secondary flow patterns that develop downstream from a bifurcation. Schroter and Sudlow[9] identified these flows as occurring both on inspiration and expiration. On inspiration a pair of vortices develop in each daughter branch. They are strong enough to complete one helical cycle within three diameters downstream. Under this condition, by the rotation of the fibers, the impaction efficiency is expected to drop significantly. In the experiments reported here, the enhancement of interception and sedimentation is not expected to play an important role, since the contribution of these mechanisms to deposition is relatively low. In contrast any change in the impaction efficiency would be fully reflected on the observations. In addition to the secondary flows developed during inhalation, on expiration a set of four vortices are generated in the parent airway ahead of the bifurcation. The effect of this air pattern on deposition has not been investigated. However, it is reasonable to assume that, using the rationale suggested for the secondary airflow patterns which develop on inspiration, deposition would be further enhanced by interception and sedimentation, and further diminished by impaction. It should be noted that no attempt was made to control the branching angle of the bronchi in this study. The angle is fixed by the rigid cartilaginous structure for the first 0.5–1.0 cm from the carinal ridge. Estimates of the branching angle in these calf lungs appeared to correlate well with reported branching angles in the human lung. Thus the use of the Stokes' number for fibers and compact particles in the estimation of impactive deposition in the human lung should be reasonable.

## CONCLUSIONS

Mathematical modeling, animal exposure, airway simulator and human exposure experiments have been employed to predict the deposition of compact particles in the human respiratory system. The depositional probabilities of spheres is modeled by relation to an associated aerodynamic equivalent diameter with reasonable accuracy. In the estimation of impaction potential fibers, Figure 3 suggests that there is no discernible difference in the deposition of fibers and spheres as a function of Stokes number when the actual diameter is employed for the spheres and the empirical impaction diameter is employed for the fibers.

This implies that not only can the impaction diameter be employed as a viable predictor of impaction, but one may estimate the series of fiber parameters that comprise the smallest fibers removable in the lung by this mechanism. That is to say that for every fiber diameter there will correspond a length that will represent the smallest fiber of that diameter that will be removed by impaction early on.

Weibel[10] has provided an exhaustive description of lung architecture developed from airway casts. Using his information and assuming that given a particle size, shape and density impaction is governed only by the airway radius and average conveyance velocity, an impaction index may be calculated as a ratio of the average velocity to airway radius. Such an impaction index for the first ten generations of human respiratory tract with liter/sec airflow is given in Table II. Clearly this potential reaches its maximum in the third through sixth generation with the fifth generation theoretically possessing the largest capability. We may visualize the first five generations as successive impaction stages, each stage being capable of removing successively smaller particle sizes. The lower limit of removal by this mechanism is then related to the 5 characteristics of the final stage. For instance, a unit density fiber 4.4 μm in diameter

Table II

Impaction Index for the First Ten Generations in the Human Lung

| Generation | Velocity cm/sec | Radius cm | Impaction Index 1/sec |
|---|---|---|---|
| 0 (trachea) | 393 | 0.900 | 437 |
| 1 | 427 | 0.610 | 700 |
| 2 | 462 | 0.415 | 1113 |
| 3 | 507 | 0.280 | 1810 |
| 4 | 392 | 0.225 | 1742 |
| 5 | 325 | 0.175 | 1857 |
| 6 | 254 | 0.140 | 1814 |
| 7 | 188 | 0.115 | 1634 |
| 8 | 144 | 0.093 | 1548 |
| 9 | 105 | 0.077 | 1364 |
| 10 | 74 | 0.065 | 1138 |

would have to be almost 90 µm long to afford complete removal. The fiber size parameters decrease with an increase in the tidal volume. Thus, at a 1450 cc tidal volume, the fiber would only have to possess an impaction diameter of 10.2 µm. This criteria would be satisfied by a unit density fiber 4.1 µm in diameter and 82 µm in length. If we further assume a fiber to have a density equal to 2.5 gm/cc (asbestos or fiberglass), the lower size limit for impaction in the lung (T.V. = 1450 cc) becomes a 3.6 × 29 µm fiber. Gross et al.[11] has observed that fibers present in the lungs of fibrous glass workers at autopsy are rarely in excess of 3.5 µm in diameter with an average length of 27 µm. This suggests that impaction initially prescreens those fiber sizes within the first six or so generations effectively and those fibers which pass this prescreening are then available for removal by sedimentation, diffusion and interception in the finer airways.

The method presented for studying particle deposition at the carina in excised lungs can be utilized to investigate the influence of sedimentation, interception and diffusion on fiber removal. Altering the physical properties of the particles and/or the tracheal velocities should provide suitable conditions to derive empirical relationships defining the interplay of particle and airflow characteristics and removal efficiencies via these other deposition mechanisms. For example, by choosing several large fiber sizes of equal impactive potential but of dramatically different length, the deposition enhancement by interception may be investigated.

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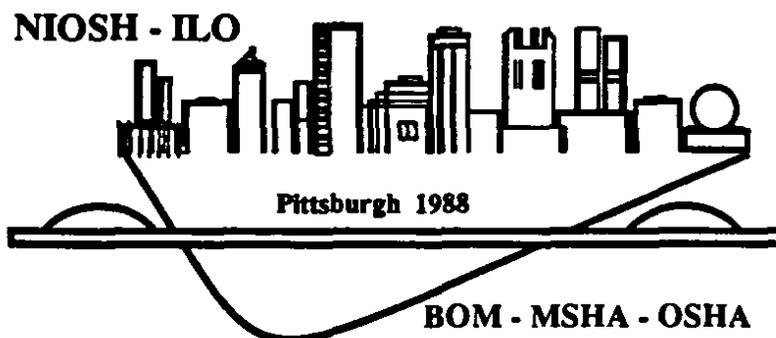
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