Pneumonectomy results in a large residual space full of an ideal culture medium of blood and fluid which, if infected, poses a tricky therapeutic problem. Various methods have been recommended for the management of this complication.14 Rosenfeldt et al13 have successfully used a technique of cyclic irrigation with antibiotic or antiseptic solution. In our case, the organism grown was resistant to all antibiotics. The patient was adamant against conventional chest drainage. Under the circumstances we considered povidone iodine to be the antiseptic solution of choice for topical use in view of its lack of toxicity.3 We tried a very simplified technique of irrigation of the infected post-pneumonectomy pleural space for the first time with povidone iodine and it proved to be successful.

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**Sexual Dysfunction, Erectile Impotence and Obstructive Azospermia in Respiratory Disease**

**Relevance of Lung-mediated Regulation of Prostaglandins**

*To the Editor:* It has been reported that more than 50 percent of men with primary azospermia have respiratory tract defects including bronchitis, bronchiectasis and sinusitis. Sexual dysfunction and erectile impotence have been reported to occur in chronic obstructive pulmonary disease. I suggest that lung-mediated regulation of prostaglandins could explain the sexual dysfunction and azospermia.

Dihomogamagamolinonic acid (DGLA) gives rise to prostaglandins (PGs) of the 1-series, notably PGE, Arachidonic acid produces PGs of the 2-series including PGE, PGE, prostacyclin and the related thromboxane A,. There seems to be a reciprocal relationship between the 1-series PGs derived from DGLA and the 2-series PGs derived from arachidonic acid. PGE, by regulating cyclic AMP formation, keeps production of the 2-series PGs in check by inhibiting the enzyme which mobilizes arachidonic acid from its stores in cell membranes.

The lungs regulate concentration of PGs reaching systemic circulation and protect the organism from excessive levels of PGs.4 PG release in the lungs is increased after hyperventilation, mechanical stimulation and respiratory alkalosis5 and increased by hypoxia.6 Many agents increase the plasma and tissue concentrations of PGs by inhibiting the activity of prostaglandin dehydrogenase and prostaglandin reductase which are responsible for their metabolic inactivation.4 PGE, is known to cause a decrease in sperm count and cause inhibition of spermatogenesis8 and to lower testosterone levels.10

A recent review on aspects of male reproductive pharmacology and toxicology dealt with calcium ion movement into the cell in maintaining acrosome stability and the blood-testis barrier to testicular toxicity.9 Prostaglandins are involved in the regulation of calcium at the cell membrane and probably in the regulation of permeability factors affecting the blood-testis barrier since PGE, prevents rises in vascular permeability due to immune complexes. C3a, histamine and serotonin.11

Thus, it is tempting to suggest that perhaps due to respiratory disease, metabolic inactivation of PGE, is incomplete allowing PGE, to enter as a systemic circulating hormone lowering PGE, in the process and directly or indirectly affecting sexual function and/or spermatogenesis.

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**Erratum**

*To the Editor:* When we were correcting the galley proofs of our recent article entitled "The Accuracy and Response Characteristics of a Simplified Ear Oximeter" (Chest 1983; 83:860-64), we caused an inadvertent error in the typesetting. The authors should be listed as K. R. Chapman, M.D.; A. D’Urzo, B.Ph.E.; and A. S. Rebuck, M.D., F.C.C.P.

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