

## SECOND HAND ROSE

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*It's no wonder that I feel abused, I never get a thing that ain't been used. Everyone knows, that's why they call me Second hand Rose" - So sang Barbara Streisand in 1965, lamenting everything from her used toothpicks, to hand-me-down hats and clothes, to someone else's discarded baby-grand piano. Second Hand Rose - It was a song that pleads "I am better than that! Why is this all I get?"*

And the same might be said about respiratory therapists, and other healthcare practitioners -- but particularly respiratory therapists -- who currently have little choice but to breathe second-hand aerosols, whether they like it or not, every day that they are on the job. So what, you might say? I spend a good part of my day giving albuterol treatments. How bad could that be? Anyway, albuterol's good stuff, isn't it? And besides, how much of that stuff do I really inhale anyway? What the hell; if it's good for the patients, it ought to be alright for me. Right?

Well, not exactly. I am not sure, but I have this uneasy feeling that chronically breathing second hand medication aerosols is a ticking time bomb that is eventually going to blow. And when it does, it is going to take many respiratory therapists with it. Maybe it has already. Toxicologists look at what they call the "dose/duration" relationship. The risk of low-dose/long-term exposure to a potentially toxic agent is often as great, if not greater, than high-dose/short-term exposures. The facts are that, at the present time, there are no facts in this area. There are

numerous well known risks to being a practicing hospital-based respiratory therapist, and if you've been in the field for more than a year, you have probably already encountered most of them. There exists the slight possibility of slipping and spraining your ankle while running to a Code. There exists the possibility of physical injury from lifting or pushing heavy patients or equipment. There exists the very serious possibility of needle-stick injury and concomitant exposure to a variety of blood-borne infections of known repute. And there exists the equally serious possibility of contracting tuberculosis, influenza, pneumonia, or just a plain old bad cold or URI. The mitigation of these risks in most hospitals is part of ongoing programs, usually administered by Infection Control, Risk Management, Employee Health and related departments. We're admonished and scolded if we are caught running in the hallways -- even for a Code. We receive training in body mechanics and proper lifting/pushing procedures. We even have state laws, in many places, regarding procedures for avoiding blood-borne infections as well as similar laws plus local policies and procedures for protecting against air-borne transmission of disease. But we do not have widespread provisions for protecting respiratory therapists against the one and only fundamental risk they routinely encounter almost constantly: inhalation of second-hand aerosol medications and exhaled patient droplets. To be fair, the risks are, at best, theoretical. But they are also, nonetheless, probable. Just because we are not holding the "smoking gun," does not mean it does not exist.

I think most of us who have been in the field for more years than we like to admit will probably recall the precautions that were taken back in the days when we routinely administered pentamidine by small volume nebulizer to HIV+ patients, and ribavirin by the so-called SPAG to bronchiolitic infants in hoods. There seemed to be a plurality of concern at that time about exposure to those agents. We (RTs) were apprehensive, the nurses were frightened, the drug companies were concerned, the aerosol device companies were concerned and, importantly, the authorities in the organizations for which we worked were also concerned. We are no longer using those medications as frequently as we once were, so the anxiety has justifiably waned. But I want to know why there does not seem to be any contemporary concern about the vast amount of albuterol aerosol that respiratory therapists must certainly inhale on the job? If you are a respiratory therapist, and are not at least wondering about this, why not?

The question "Does aerosol therapy place healthcare workers at risk of adverse occupational exposure?" is the root question that needs to be answered. Unfortunately, only a few meager efforts at answering this question have even been attempted. Let us briefly examine those reports. Kern and co-workers reported in 1989 that in a survey study of 194 RTs and 517 controls, there was a previously unrecognized excess of asthma among RTs. The excess develops after entry into the profession and does not appear to be explained by confounding, information bias or selection bias. In 1993 Christiani and Kern published results of

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an epidemiological study of 2,086 RTs and 2,030 physical therapists surveyed about a history of physician-diagnosed asthma. Analysis of practitioners who developed asthma after entry into their profession revealed that RTs had a significantly greater risk, 7.4 versus 2.8%, than physical therapists. The odds ratio for the RT was 2.5 (95% CI, 1.6 to 3.3) after adjustment for age, family history, atopic history, smoking, and gender. The authors speculated that this could be due to occupational exposure and suggested that identification of potential responsible agents should be sought. Carnathan et al reported in 2001 that both isomers (R-albuterol and S-albuterol) of racemic albuterol (RAC) are detectable in the plasma of RTs after occupational exposure to nebulized RAC given to patients. The proinflammatory S-isomer achieved higher plasma levels, remained in the circulation and seemed to correlate with duration of exposure. The title of the Carnathan paper, incidentally, is the first instance I can find of the use of the apt term "Second Hand Albuterol."

In 2003 Jones and co-workers from the United Kingdom published a study that examined the presence of epidemic strains of *Pseudomonas aeruginosa* in the environment of a Cystic Fibrosis treatment center during a cross-infection outbreak. Microbiological sampling of the environment was conducted along with bacterial fingerprinting to compare against the typing patterns of bacteria responsible for cross-infection of patients. They isolated epidemic *P. aeruginosa* strains from room air when patients performed spirometry, airway clearance and nebulization procedures. They concluded that aerosol dissemination may

be the most important factor in patient-to-patient cross-infection outbreaks. Similarly, in their 2003 paper describing the initial outbreak of SARS in Hong Kong, Lee and colleagues stated "We suspected that the infection was transmitted by droplets and possibly by fomites, and we therefore instituted both airborne precautions (e.g., use of the N-95 respiratory) and contact precautions (e.g., use of gown and gloves), as recommended by the CDC. However, the use of a jet nebulizer to administer aerosolized albuterol in the index patient had probably aggravated the spread of the disease by droplet infections." By the way, half of the 138 patients discussed in this report were health care workers. In a 2004 publication by Somogyi and co-workers in Canada, a vivid plume of exhaled aerosol droplets, emanating from the exhalation ports of a mask on a subject receiving an aerosol treatment, is dramatically demonstrated by photography.

There is also emerging apprehension about the downregulation of beta 2-adrenergic receptors as a result of chronic exposure to beta agonists, the most common of which, of course, is albuterol. A pharmacological study by Witt-Enderby and associates in 1993 begins with the concern that "Scheduled chronic administration of beta 2-adrenoceptor agonist bronchodilators in patients with asthma recently has been reported to be associated with a worsening of symptoms and an increase in bronchial responsiveness." The investigators studied the bronchial responsiveness in vitro in a group of rabbit airways subjected to a 28 day in vivo exposure to albuterol. The maximum contraction response to methacholine was significantly increased in the albuterol-treated group as compared to an untreated control group. Granted, this is an animal study on the laboratory bench, but it raises the possibility that chronic exposure to albuterol results in a paradoxical increase in bronchoreactivity.

I believe that protecting the professional respiratory therapist ought to be given new priority in the workplace. There may be a two-pronged risk to the respiratory therapist during aerosol therapy. First, there's the risk of inhaling drugs that are not prescribed for the practitioner and which could gradually be inhaled in potentially toxic amounts (in view of the chronic dose-duration exposure relationship). The source of these aerosol drugs is not only from direct patient-exhalation (nebulizers, MDIs, DPIs) but from aerosol delivery devices, such as liquid nebulizers that, by design, vent large amounts of aerosol into the environment during the patient's exhalation phase. High-flow large volume continuous nebulizers likewise vent large quantities of medication into the ambient air because they provide much more flow than patients actually require for all but a small fraction of the inspiratory phase of the respiratory cycle. Second, there's a potential risk of inhaling infectious droplets during aerosol therapy, particularly from patients with a respiratory infection that has not yet been diagnosed. These risks may exemplify sound reasons why non-indicated aerosol therapy clearly should not be given. But, to the extent that aerosol therapy IS indicated, clearly a great deal more investigation needs to be done. Part of that investigation also needs to focus on the effect of occupational exposure to albuterol and other medications on the health care practitioners who are most directly in the line of fire of the contemporary smoking gun we call a nebulizer.

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