The Impact of Substance Abuse on the Respiratory System*

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Substance abuse has become pervasive in our society. The National Institute on Drug Abuse estimates that there are 800,000 heroin users, five million regular cocaine users, and 10 million regular marijuana users. Numerous studies have indicated that between 15 and 40 percent of patients hospitalized in a general hospital are active abusers of alcohol or other drugs and are hospitalized because of problems associated with the abuse. With this high level of abuse and the need for medical care in this population, it is the rare health practitioner who does not encounter some patients who abuse illicit or prescription drugs.

The respiratory system is invariably exposed to these drugs and is affected by them, either directly or secondarily, on a temporary or permanent basis. This review will examine the range of responses that can be encountered in the respiratory systems of substance abusers. Since the practitioner is usually confronted with a complaint or clinical syndrome, this review is organized according to clinical manifestations. Table 1 enables the reader to quickly determine which patterns of respiratory involvement are associated with a particular type of substance abuse. Because tobacco and alcohol have been extensively reviewed elsewhere,14 they will not be dealt with here. Similarly, management will be discussed only when unique aspects pertain.

General Consideration: Styles of Drug Abuse

The patterns or types of substance abuse that are prevalent in a given patient population are, in part, determined by a variety of external factors such as the cost or availability of particular substances with abuse potential, peer pressure, local or group fads, and legal pressures. The potential for respiratory system complications is, in turn, dependent not only on the particular substance abused but also on the route of administration, the origin of the abused substance, contaminants present in the dry sample, whether or not the pattern of use involves sharing of paraphernalia, and the host responses of the individual user. Practitioners must remain aware of local trends in abuse patterns. Publications such as the Drug Abuse Warning Network (DAWN) of the National Institute of Drug Abuse and the Morbidity and Mortality Weekly Report may be quite useful in this regard.

Infectious Respiratory Complications

Perhaps the most familiar respiratory system complication of substance abuse is lung abscesses. Abscesses may be localized to a single area,2 in which case they are invariably related to aspiration during a stupor induced by narcotics or sedative hypnotics, or there may be sequentially appearing multiple abscesses. In the latter case, intravenous (mainlining) or occasionally intradermal injections (skin popping) produce right sided endocarditis or septic thrombophlebitis, most commonly with Staphylococcus aureus, Candida species or, occasionally, Gram-negative organisms.

A variety of other, less well known, pulmonary infections may also result from substance abuse. Reichman and associates5 documented an increased risk of both pulmonary and extrapulmonary forms of tuberculosis among heroin addicts. The contamination of marijuana by various fungi and the presence of precipitins to fungi in the blood of smokers9 suggests that exposure to fungal material may be found among marijuana smokers. A case report in which contamination of marijuana with Aspergillus fumigatus led to infection and pneumonitis in a young man with chronic granulomatous disease4 and another report in which the smoking of moldy marijuana was associated with the development of allergic bronchopulmonary aspergillosis2 indicates that this is a possible route of lung infection in susceptible persons. Fungal contamination

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Table 1—Forms of Substance Abuse and the Associated Patterns of Respiratory Complications

<table>
<thead>
<tr>
<th>Substance Class*</th>
<th>Specific Agents</th>
<th>Route</th>
<th>Respiratory Complication</th>
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<tbody>
<tr>
<td>Sedatives/tranquilizers</td>
<td>Ethchlorvynol</td>
<td>IV</td>
<td>Respiratory depression&lt;sup&gt;45,46&lt;/sup&gt;</td>
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<td></td>
<td>Diazepam, Chlordiazepoxide</td>
<td>PO</td>
<td>Pulmonary edema&lt;sup&gt;45-47&lt;/sup&gt;</td>
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<td></td>
<td>Glutethimide</td>
<td></td>
<td>Aspiration pneumonia&lt;sup&gt;46&lt;/sup&gt;</td>
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<td></td>
<td>Paraldehyde</td>
<td></td>
<td>Atelectasis&lt;sup&gt;44&lt;/sup&gt;</td>
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<td></td>
<td>Phenobarbitol</td>
<td></td>
<td>Drug interaction (esp phenobarb)</td>
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<tr>
<td>Stimulants</td>
<td>Methylphenidate</td>
<td>PO</td>
<td>Abnormalities of DCO&lt;sup&gt;39&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>Cocaine</td>
<td>smoke</td>
<td>AIDS with pul infections&lt;sup&gt;4&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>Amphetamines</td>
<td>snorted</td>
<td>Broncholiths obliterans/organizing pneumonia&lt;sup&gt;4&lt;/sup&gt;</td>
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<td>Narcotics</td>
<td>Heroin</td>
<td>Subcutaneous</td>
<td>Granulomatous pneumonitis&lt;sup&gt;27,29&lt;/sup&gt;</td>
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<td></td>
<td>Methadone</td>
<td>IV</td>
<td>Nasal septal perforation&lt;sup&gt;17&lt;/sup&gt;</td>
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<td></td>
<td>Pentazocine</td>
<td>PO</td>
<td>Pneumomediastinum&lt;sup&gt;20&lt;/sup&gt;</td>
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<td></td>
<td>Codeine</td>
<td>Smoked</td>
<td>Pulmonary edema&lt;sup&gt;48&lt;/sup&gt;</td>
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<td>Propoxyphene</td>
<td>Snorted</td>
<td>Pulmonary hypertension&lt;sup&gt;34,47&lt;/sup&gt;</td>
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<td>Cannabinoids</td>
<td>Marijuana</td>
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<td>AIDS with pul infections&lt;sup&gt;4&lt;/sup&gt;</td>
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<td></td>
<td>Hashish</td>
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<td>Airways obstruction&lt;sup&gt;32,43,46&lt;/sup&gt;</td>
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<td>Aspiration pneumonia&lt;sup&gt;4&lt;/sup&gt;</td>
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<td>Cannabinoids</td>
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<td>Bronchiectasis&lt;sup&gt;19&lt;/sup&gt;</td>
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<td>Bullous damage&lt;sup&gt;43&lt;/sup&gt;</td>
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<td>Diffusing capacity/gas exchange abnormalities&lt;sup&gt;34,46,49&lt;/sup&gt;</td>
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<td>Fibrosis/granulomatosis&lt;sup&gt;30,34,41&lt;/sup&gt;</td>
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<td>Infections&lt;sup&gt;41&lt;/sup&gt;</td>
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<td>Mediastinal/hilar adenopathy&lt;sup&gt;27,50&lt;/sup&gt;</td>
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<td>Nasal septal perforation&lt;sup&gt;17&lt;/sup&gt;</td>
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<td>Pneumomediastinum&lt;sup&gt;39&lt;/sup&gt;</td>
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<td>Pulmonary edema&lt;sup&gt;48&lt;/sup&gt;</td>
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<td>Respiratory depression&lt;sup&gt;39,40&lt;/sup&gt;</td>
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<td>Vascular involvement&lt;sup&gt;30,34,41&lt;/sup&gt;</td>
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<td>Volatile hydrocarbons</td>
<td>Glue</td>
<td>Inhaled</td>
<td>Bronchodilatation (acute)&lt;sup&gt;11-13&lt;/sup&gt;</td>
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<td>Paint thinner</td>
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<td>Bronchoconstriction (chronic)&lt;sup&gt;14,15&lt;/sup&gt;</td>
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<td></td>
<td>Butane</td>
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<td>Bronchitis&lt;sup&gt;18&lt;/sup&gt;</td>
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<td>Candidiasis&lt;sup&gt;16&lt;/sup&gt;</td>
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<td>Pharyngitis/rhinitis&lt;sup&gt;48&lt;/sup&gt;</td>
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<td>Reduction in diffusing capacity&lt;sup&gt;51&lt;/sup&gt;</td>
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<td>rate theophylline clearance&lt;sup&gt;46&lt;/sup&gt;</td>
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<td>Pulmonary fibrosis&lt;sup&gt;20&lt;/sup&gt;</td>
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<td>Nasal rash&lt;sup&gt;20&lt;/sup&gt;</td>
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<td>Laryngospasm&lt;sup&gt;20,30&lt;/sup&gt;</td>
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<td></td>
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<td>Pulmonary edema&lt;sup&gt;49&lt;/sup&gt;</td>
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*The complications listed are representative of those reported in the medical literature. Although a particular complication may not have been associated with the use of all agents in a class substance, it is presumed that these complications may occur with the use of other agents in the same substance class. (?) indicates a particularly tenuous association.

of heroin and the high prevalence of serum precipitins in heroin users<sup>4</sup> suggests that this form of abuse may also be a vehicle for infection.

More recently, the Acquired Immune Deficiency Syndrome (AIDS), with its myriad of pulmonary infectious complications, has been thoroughly described among abusers who inject drugs intravenously.<sup>4</sup>

**AIRWAYS COMPLICATIONS**

A variety of abused materials may involve the airway. This site of involvement can occur after parenteral injection as well as following inhalation. Although an unusually high prevalence of airways obstruction seems to occur among a variety of drug users, a causal relationship has not been established. Banner and co-workers<sup>10</sup> have described symptomatic bronchiectasis in heroin addicts with known prior episodes of heroin-induced pulmonary edema. Bronchography, performed months after the resolution of acute edema, showed either diffuse or lower lobe involvement in these subjects. The mechanism of bronchial injury is uncertain although simple aspiration during a period of stupor appears unlikely.

Allergic bronchopulmonary aspergillosis (ABPA) has been reported in an atopic marijuana smoker whose stock of marijuana yielded a heavy growth of several...
species of Aspergillus. Marijuana's other effects on the airways have received considerable attention because of the drug's putative action as a bronchodilator. Vachon and associates14 reported up to a 38 percent mean reduction in airway resistance (Raw) in a group of normal volunteers with prior marijuana smoking experience who smoked marijuana with a high delta-9 tetrahydrocannabinol (THC) concentration. Lesser reductions in mean airway resistance were obtained without accompanying tachycardia by smoking marijuana with lower concentrations of THC. These observations were extended to clinically stable asthmatic subjects by Tashkin and co-workers15 who demonstrated, in a double-blind placebo controlled study, that both smoked marijuana and oral THC could reduce Raw for up to several hours.16 This bronchodilator effect was shown to be independent of antimuscarinic or beta agonist activity.13 Despite the bronchodilator effect of smoked marijuana in the acute setting, heavy use over 47 to 59 days by normal subjects was associated with mild but significant reduction in expired volume in one second, maximum mid-expiratory flow (MMEF), and specific airway conductance.14 These changes were presumed to be due to the chronic irritant effect of the marijuana smoke. Furthermore, chronic studies suggest that at least a partial tolerance to the acute pharmacologic bronchodilator effect occurs in chronic users.14 Reduction in specific airway conductance, not detectable in age-matched tobacco smokers, have also been demonstrated in habitual marijuana smokers.15 The clinical significance of these findings remains uncertain.

Smoking another derivative of the Cannabis plant, hashish, has been associated with a variety of upper and lower respiratory tract ailments. Henderson and co-workers16 studied a group of 200 young soldiers. Seventy-five percent had pharyngitis that was rarely associated with oral candidiasis. Hashish use by these individuals was usually moderate (ie, less than 25 g monthly). The remaining 25 percent smoked more than 50 g of hashish monthly, and they had symptoms of rhinitis or chronic bronchitis. Bronchoscopy was performed in six individuals. All had bronchial mucosal injection, mucopurulent exudates, and inflammatory changes in biopsy specimens of mucosa. Although this was an uncontrolled study, cigarette smoking did not appear to account for the observed abnormalities.

Bronchiolitis obliterans and respiratory failure have been associated with smoking of freebase cocaine (P. Hopewell, personal communication). In another patient, the syndrome of bronchiolitis obliterans with organizing pneumonia was diagnosed in a freebase user. Treatment with high doses of corticosteroids produced sufficient improvement to allow extubation and weaning from supplemental oxygen. However, dyspnea on exertion, airways obstruction, and a reduction in diffusing capacity (Dco) were present eight months after the acute illness (S. Schonfeld, personal communication). The relationship between bronchiolitis and freebase cocaine remains to be defined.

Injury to the upper airway has been well described following the sniffing of various materials. Nasal septal inflammation and perforation are most commonly associated with the use of cocaine but may actually occur only rarely in that setting.17 Less commonly appreciated is the fact that septal injury has been reported in up to 5 percent of heroin users.18 Perforations associated with drug snorting can occur anywhere along the septum depending upon the technique used to sniff the drug. Perforations may be more prevalent among women. Although these lesions appear to be benign even when they are quite large, they do not appear to regress with cessation of drug snorting. The presence of such a lesion should alert the clinician to the possibility of substance abuse despite the absence of other stigmata. Inhalation of volatile hydrocarbons like glue or paint thinner may produce a rash around the nose and mouth in habitual users.19 Aerosol sniffing has been associated with laryngospasm and death, although cardiac arrhythmias may play a more important role.20

PARENCHYMAL ABNORMALITIES

Pulmonary Edema

Probably the best described pulmonary complication of substance abuse is pulmonary edema. The adult respiratory distress syndrome and lesser degrees of noncardiogenic lung edema occur in association with virtually all routes of administration and most classes of illicitly used materials including narcotics24-25 and other analgesics,24 sedative hypnotics,25-27 cocaine,28 and hydrocarbons.29,30 Although the mechanism underlying the edema remains unknown, the edema fluid protein concentration approaches serum levels as in most types of the adult respiratory distress syndrome.28,30 In an autopsy study of 17 intravenous drug abusers of whom eight died from acute narcotic, acute alveolitis consisting primarily of neutrophils with occasional eosinophils and mononuclear cells was commonly seen. Frank edema was also common, particularly in the subgroup dying from a drug overdose. The relationship of the alveolitis to the development of edema was unclear.31

The onset of pulmonary edema following ingestion varies with the substance used and the route of administration but usually occurs within the first several hours following use. However, the onset of edema may be delayed up to 24 hours.32 Clinically and roentgenographically, there is little to distinguish these patients from any other patients with noncardiogenic pulmonary edema unrelated to substance
abuse. Pulmonary edema related to most types of substance abuse occurs in relatively young individuals. Stigmata of parenteral administration may be apparent when that route has been used. Since many instances of pulmonary edema are related to overdoses of sedative drugs, stupor or coma may initially be present. However, this is not invariably the case, and the absence of an alteration in the level of consciousness by no means excludes substance abuse as the cause of the edema. Because there is frequently associated ventilatory depression, these patients may show profound respiratory acidosis, as well as a concomitant metabolic acidosis. Aspiration is a serious threat in stuporous patients and may contribute to the pulmonary edema. Management of these patients is supportive. In cases of opioid abuse, naloxone can be given to improve level of consciousness and ventilatory drive. This may obviate the need for intubation and mechanical ventilation. The edema may resolve over a period of hours, or, when complicated by aspiration, may persist for some weeks. Physiologic improvement follows a similar time course with generally rapid improvement in arterial blood gas levels. As with most types of noncardiogenic edema, several weeks are usually required before lung volumes, compliance, and diffusing capacity attain maximal levels.

Pulmonary Interstitial Reactions

Pulmonary fibrosis, often with associated granuloma formation, has been most frequently described in relation to the intravenous injection of drugs containing particulate materials, especially talc. However, it can occur via other routes of administration as exemplified by the smoking of paraquat treated marijuana. Arnett and co-workers described two histologic patterns of granulomatosis resulting from intravenous injection of talc containing drugs intended for oral use. When the granulomas were concentrated predominantly within pulmonary arteries, pulmonary hypertension was common. Among subjects without pulmonary hypertension, granuloma formation occurred predominantly within the interstitium. It is not clear why some persons develop interstitial granulomas while others develop angiocentric lesions in response to the same material. It does appear, however, that the filler substance rather than the actual drugs injected is the inciting agent for this type of reaction. Thus, the intravenous injection of oral preparations containing talc or methylcellulose as a mordant are most likely to be associated with these types of granulomatoses. In a study of 17 intravenous abusers of crushed methadone tablets, dyspnea, at times severe, was common. The diffusing capacity was reduced in ten and chest x-ray films were distinctly abnormal in seven subjects. A pattern of widespread fine nodular densities, with or without associated volume loss was seen on the chest x-ray films. There was a tendency for the densities to increase in size with the passage of time. Of interest, nine of fifteen subjects studied had evidence of tcalc retinopathy on fundoscopic examination that appeared as small white glistening dots concentrated about the macula. Patients with the most advanced retinal involvement also have small hemorrhages about the macula. Lung histology was available from one of these 17 patients and revealed severe pulmonary fibrosis and interstitial granulomatosis, some areas of which contained talc crystals. Others have reported the development of large conglomerate mass lesions evolving over a period of months to several years among persons injecting crushed methadone tablets. The mechanisms underlying granuloma formation and fibrosis remain obscure. Although granulomas in some individuals may present hypersensitivity reactions to fungal antigens contaminating the abused material, this is probably a rare occurrence. Bronchoalveolar lavage (BAL) from IV pentazocine-abusing patients contains an increased number of cells, almost all macrophages. The type and function of lung and peripheral blood lymphocytes appear normal. This suggests that the underlying process differs from other fibrosing and granuloma producing conditions such as hypersensitivity pneumonitis, sarcoidosis and asbestosis. It should be noted, however, that there are similarities to sarcoid and that among persons injecting crushed pentazocine tablets pneumonitis with an increase in BAL and serum angiotensin converting enzyme (ACE) levels and increased 67Ga uptake in the lungs has been reported. The BAL also contained talc-like particles.

Bullous Parenchymal Changes

Recently, the occurrence of bullous pulmonary damage was reported among intravenous drug abusers. The precise material injected was not known. Bullae were large and were confined to the upper lobes. They were associated with moderate to severe airflow obstruction and reduction in single breath carbon monoxide diffusing capacity. Although all the drug abusers reported smoking cigarettes, their average age (36.7 years) was a decade younger than that of a comparison group of patients with more diffuse bullous changes and no history of intravenous drug use. The mechanism of bullous damage among these intravenous drug users is not known but the authors of the report speculated that repeated damage to the pulmonary capillary bed might result in lung damage and bullae formation.

Atelectasis

Atelectasis may develop in persons abusing a wide variety of substances that cause respiratory depression or predispose to the retention of secretions.
degree of involvement can range from segmental atelectasis to collapse of an entire lung. This problem is frequently asymptomatic.

**Pulmonary Vascular Complications**

Pulmonary vascular abnormalities occur following abuse of a number of drugs via various routes of administration, and may range from minimal, barely detectable physiologic abnormalities to severe pulmonary hypertension. Reductions in regional perfusion and ventilation as determined by lung scan have been reported in asymptomatic heroin addicts. The ventilation abnormalities resolved with cessation of heroin use whereas the perfusion defects have persisted. Embolization of particulate matter to pulmonary vessels with subsequent granuloma formation was postulated as the cause of the perfusion abnormalities.\(^{45}\) Angiothrombotic lesions, including vascular webs and plexiform lesions, have been associated with the intravenous injection of oral preparations.\(^{39}\) Foreign body granulomas have been found in persons who use a variety of preparations intravenously.\(^{35,36,47}\) Symptoms of dyspnea and advanced pulmonary hypertension develop in some of them. As noted previously, persons with pulmonary hypertension typically have evidence of granuloma formation within the pulmonary vessels.\(^{38}\) As might be expected, most of these patients also have substantial reductions in carbon monoxide diffusing capacity. Abnormalities of the diffusing capacity have been reported in intravenous users of heroin and methadone,\(^{36,48}\) talwin, and pyribenzamine\(^{49}\) and among persons using amphetamine preparations intended for nasal spraying.\(^{47}\) We observed similar abnormalities among smokers of freebase cocaine.\(^{50}\) More recently, significant reductions in diffusing capacity have been reported among chronic heavy marijuana and tobacco smokers.\(^{51}\) The prevalence of alterations in diffusing capacity and the extent to which such changes reflect pulmonary vascular rather than parenchymal involvement is difficult to define. Although these physiologic abnormalities are frequently associated with symptoms of dyspnea and cough, symptoms are not invariable. Among heroin abusers, for example, up to half manifest a reduction in DCO, usually without associated symptoms.\(^{52}\) Our experience suggests that among populations intravenously abusing material with a high particulate content, there is a greater potential for vascular involvement, even after relatively brief periods of abuse. Indeed, it appears that the particulate concentration rather than the nature of the substance used is the major factor responsible for the pattern and severity of pulmonary vascular injury in IV drug abusers. Among those favoring other routes, the vasoactive properties of the substance used may determine the likelihood of vascular complications.\(^{53}\)

**Pleural and Mediastinal Complications**

Pleural and mediastinal complications of substance abuse are relatively uncommon. Pleural abnormalities are most likely to arise as an extension of some underlying parenchymal process. Septic emboli may occur as a complication of tricuspid endocarditis, thrombophlebitis, or cellulitis in parenteral drug abusers. On occasion, these emboli may involve the pleural space in the form of empyema, pneumothorax, or bronchopleural fistula. A post mortem report described pleural studding by talc granulomata. These nodules were 1 to 3 mm in size and contained birefringent crystals.\(^{48}\) Pleural effusions have not been noted as a part of drug induced pulmonary edema in the absence of additional complications.

Pneumomediastinum, rarely with pneumothorax, has been associated with freebase cocaine smoking,\(^{59}\) marijuana smoking, and the intravenous injection of heroin.\(^{54,55}\) In each patient, a prolonged Valsalva maneuver was described as a part of the ritual by which the affected subjects used their drugs. Other causes of pneumomediastinum were excluded. In most instances, these patients were asymptomatic with respect to the mediastinal air and the process resolved, untreated, over a period of several days.

Hilar and mediastinal adenopathy, in association with the intravenous use of talc-containing substances, are quite exceptional.\(^{35,50}\) Recently, four patients, including three intravenous drug users, with septic pulmonary emboli due to \textit{S} aureus were described as having hilar and mediastinal adenopathy that resolved with antibiotic therapy.\(^{57}\) Similar findings were described in a drug abuser with right-sided endocarditis in the absence of pulmonary infiltrates.\(^{58}\) The frequency of this phenomenon is unknown.

**Control of Ventilation**

Overdose with narcotics, tranquilizers, and many if not all sedative hypnotics is well recognized as a cause of acute ventilatory failure. The implications of lesser degrees of alteration in ventilatory control which may result from the abuse of a variety of substances is less clear. Santiago and co-workers\(^{50}\) described two groups of subjects enrolled in a methadone maintenance program. In the group taking the drug less than two months, both the predose and post methadone level of arterial carbon dioxide tension were elevated and the ventilatory responses to hypoxia (and to a lesser extent carbon dioxide) were reduced as compared to a group receiving comparable doses of methadone for longer than two months. These same workers also demonstrated the apparent development of tolerance to the reduction in ventilatory compensation for increases in airway resistance that methadone and other narcotics are known to produce.\(^{59}\) Presumably, this pattern of
tolerance occurs with other narcotics. These results suggest that persons using narcotic drugs intermittently or for a relatively brief duration (i.e., less than several months) are particularly vulnerable to aberrations in ventilatory control. Although these changes in control would not be expected to produce clinical illness by themselves, it is possible that if other conditions were superimposed (e.g., bacterial pneumonia or acute bronchospasm), the resulting illness would be more severe and less responsive to usual interventions. The long range consequences of chronic ingestion of various sedative hypnotics or tranquilizers are uncertain, but a number of these agents are known to produce respiratory depression. A history of this type of chronic ingestion should be sought in patients presenting with abnormal ventilation.

DRUG INTERACTIONS

Physicians treating patients for a variety of pulmonary conditions should be alert to possible drug interactions in patients who are concomitant substance abusers. Kreek et al. reported that 21 of 30 patients developed signs of methadone withdrawal induced by rifampin. This occurred between one and 33 days after the start of rifampin therapy. Further study of these patients showed reductions of between 33 and 68 percent in the serum levels of methadone and an increase in a major hepatic metabolite of methadone. Patients receiving methadone and rifampin may require increased doses of the narcotic to compensate for the apparent enhancement of its hepatic metabolism.

Marijuana smokers, like tobacco smokers, have a reduced theophylline half-life compared to nonsmokers. Chronic daily marijuana smokers may need surprisingly large doses of theophyllines to attain therapeutic serum concentrations.

Phenobarbital has been reported to cause clinical deterioration in steroid dependent asthmatics. Phenobarbital was shown to increase the metabolic clearance rate of dexamethasone by 88 percent, thereby reducing the steroid's therapeutic effect.

CONCLUSIONS

With the increase in the number of abusers of both licit and illicit drugs, there has been a concomitant increase in the number of substance abusers seeking medical care. The medical consequences of drug abuse can be a direct effect of the drug or indirect effects due to the presence of contaminants and/or the method of administration. Effects of drug abuse on the respiratory system include infectious processes and noninfectious conditions involving the lung parenchyma, pulmonary vasculature, and pleura or mediastinum. In addition, drugs of abuse can alter the control of ventilation and may produce adverse interactions with medications prescribed for a variety of pulmonary conditions.

As the number of abusers increases and the number of abused drugs increases, the variety of complications involving the respiratory system will surely increase. Physicians should be aware of the role drug abuse can play in creating and altering clinical disease in the chest. They must consider these types of problems and carefully search for them since patients often cannot or will not indicate that they have abused drugs.

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