Spiegel der artz
ney / 60z zeypen zu nutz bnd
tröst den Leyengemachen/durch Laurentini
Friesen, aber öffent um gefolcher/durch
vnsleid der Bächercker/verund
 durch den selbigen Laurenti
enum/wiederm gebeffere
und in seiner ersten
glange ges
fetz.

Sntmit sollen wibrichs &nd falsch declariert sein/alle exemplar daß buchfs so von diesem druck aufgangen sein-

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CONTENTS

Introduction 158

Respiratory Therapy Modalities in the Treatment of Acute Respiratory Failure 159
JAMES A. L. MATHERS, JR., M.D.

Screening Pulmonary Function Tests 163
GEORGE W. BURKE, III, M.D.

Flexible Fiberoptic Bronchoscopy 169
ORHAN MUREN, M.D.

Bedside Flow-Directed Balloon Catheterization in the Critically Ill Patient 172
J. EUGENE MILLEN
FREDERICK L. GLAUSER, M.D.

Intrapulmonary Lymph Node Presenting as a 'Coin' Lesion: A Case Report
SIBU P. Saha, M.D.
PORTER MAYO, M.D.

Maxillary Periapical Actinomycosis: A Case with an Unusual Roentgenographic Appearance 178
DANNY R. SAWYER, D.D.S., PH.D.
DENNIS G. PAGE, D.D.S., M.S.

The Dental Health Status of Pre-Columbian Peruvians: A Study of Dental Caries, Missing Teeth, Attrition, Osteitis, Calculus, and Bone Loss 181
DANNY R. SAWYER, D.D.S., PH.D.
MARVIN J. ALLISON, PH.D.
RICHARD P. ELZAY, D.D.S., M.S.D.
ALEJANDRO PEZZIA, PH.D.

TABLE OF CONTENTS FOR VOLUME FOURTEEN 189

INDEX TO VOLUME FOURTEEN 191


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INTRODUCTION

The winter issue of the MCV QUARTERLY presents a departure from our usual symposium proceedings. We offer, instead, five full-length articles and two case reports that range from respiratory failure and the dental health of pre-Columbian Peruvians to a report of a rare clinical entity, an intrapulmonary lymph node presenting as a ‘coin’ lesion. We hope that our readers will find these papers interesting and informative.

THE EDITORS
Respiratory Therapy Modalities in the Treatment of Acute Respiratory Failure

JAMES A. L. MATHERS, JR., M.D.

Assistant Professor of Medicine and Co-Medical Director, Respiratory Therapy, Medical College of Virginia, Health Sciences Division of Virginia Commonwealth University, Richmond, Virginia

Rapid advances have been made in the field of respiratory therapy in the past several years, resulting in an increasing sophistication and range of application. Properly applied, these modalities have led to significantly increased survival in patients with acute respiratory failure and a decreased morbidity among individuals with chronic pulmonary insufficiency. It is the purpose of this article to put into perspective respiratory therapy techniques and their application in the treatment of acute respiratory failure. To this end, we may divide respiratory therapy into five categories: 1) oxygen delivery, 2) airway hygiene, 3) expansion therapy (lung inflation), 4) artificial airways, and 5) mechanical ventilation.

While the symptoms are not specific, the onset of acute respiratory failure is not a subtle event. Abnormal arterial blood gases are invariably present. Indeed, the entity is usually defined in terms of the degree of alteration in the arterial blood gases. Acute respiratory failure results from one of three general abnormalities: 1) neuromuscular dysfunction, 2) increased airway resistance, and 3) reduction of pulmonary compliance. In pure form each of these categories produces a predictable pattern of arterial blood gas derangement. Rational management of acute respiratory failure requires an awareness of these alterations so that the respiratory therapy may be intelligently applied to stabilize or reverse the patient’s pulmonary deterioration.

In the initial assessment of the patient in acute respiratory distress, it is important to determine the nature of the respiratory compromise. Patients with neuromuscular dysfunction such as occurs with drug overdose or Guillain-Barré syndrome may have normal lungs but do not have the ability to maintain an adequate minute ventilation. The arterial blood gas derangement consists of hypoxemia and relatively acute hypercarbia with a normal alveolar-arterial oxygen tension gradient. Respiratory failure due to a sudden increase in airway resistance includes entities such as status asthmaticus, or bronchospasm with retained secretions in patients with underlying chronic airway disease. The arterial blood gas analysis will reveal hypoxemia with hypercarbia and a widened alveolar-arterial oxygen tension gradient. The functional residual capacity is elevated and, in many patients, there is actually an increase in pulmonary compliance. The gas exchange abnormality results from ventilation-perfusion mismatching. Patients whose respiratory failure results from an acute loss of pulmonary compliance are those with an overwhelming parenchymal insult such as occurs with extensive bacterial pneumonia, aspiration, or a systemic process such as pancreatitis. These disorders may be grouped under the heading of adult respiratory distress syndrome. The physiologic alterations are hypoxemia with hypocarbia and respiratory alkalosis because of right-to-left shunting of blood, a fall in the pulmonary compliance, and a marked reduction in the functional residual capacity.
Hypoxemic Hypercapneic Respiratory Failure with Normal A-a \text{O}_2 \text{ Gradient}

The Respiratory Therapy Department of an institution becomes involved with these patients when the decision has been made to institute mechanical ventilation. While the initial selection of artificial airways is not in the domain of this department, usually it is the respiratory therapists who are called upon when these airways do not function properly. Because of the experience gained in managing airway problems, we at the Medical College of Virginia have developed a number of recommendations for this aspect of respiratory care. When selecting an artificial airway, one must make an initial decision between “compliant” and “stiff” cuffs. We prefer to use the large volume compliant cuff tubes because a seal is obtained at a lower cuff pressure. Furthermore, the intracuff pressure measured by the respiratory therapist is a more accurate assessment of pressure exerted against the tracheal wall than pressures obtained from stiff cuffs.

While orotracheal tubes may be rapidly inserted during an emergency such as a drug overdose, these tubes are associated with the highest incidence of tracheal damage since they are difficult to stabilize and have a tendency to move about in the trachea when the patient is turned, moves or coughs. Nasotracheal tubes are more stable and, when a compliant cuffed tube is placed via this route, they may be left in place for as long as two to three weeks with a minimum of tracheal damage. The cross-sectional diameter of the nasotracheal tube is smaller than the orotracheal tube. The combination of longer length and the narrow diameter may make adequate suctioning difficult. Should the management of secretions be a problem and a prolonged intubation anticipated, elective tracheostomy offers many advantages such as the wide variety of tubes available, including talking tracheostomy tubes, fenestrated tubes, and cuffless tubes; patients may be fed via the oral route; vocal cords are not damaged; and there is no damage to the nasal septum. With the advent of the compliant cuff and better management of the artificial airway, the incidence of tracheal damage has decreased significantly. It does occur from time to time, however, and we stress the importance of periodic monitoring of intracuff pressure.

When the decision has been made to intubate patients with this form of respiratory failure, and the appropriate airway has been selected, mechanical ventilation is usually instituted with relative ease because of the normal physiology of the underlying lung.

Hypoxemic Hypercapneic Respiratory Failure with Abnormal A-a \text{O}_2 \text{ Gradient}

This disturbance is most frequently found in patients with increased airway resistance due to bronchospasm, obstructing secretions, and mucosal swelling and edema. The immediate approach to the hypoxemic patient is to stabilize the arterial oxygen tension while treating the underlying defect in hopes of avoiding intubation. The goal is to maintain the \text{PaO}_2 in the range of 60 to 80 mm Hg which will insure hemoglobin saturation in excess of 90%. Further increases in the arterial oxygen tension do not significantly increase oxygen delivery to the tissues. The mode of oxygen administration is of great importance in the presence of hypercapnia. Oxygen therapy devices are divided into high-flow or low-flow systems. A high-flow apparatus mixes oxygen and room air prior to delivery to the patient. The gas flow delivered by the device is sufficient to provide the patient’s total minute ventilation. Low-flow systems do not provide enough gas flow to supply the entire inspired atmosphere, and some part of the tidal volume will be supplied by the entrainment of room air. Such systems may provide any concentration of oxygen from 21% to 95%, but the fraction of inspired oxygen (\text{F}_{\text{IO}_2}) can be altered by the patient’s respiratory pattern. Low-flow devices include the nasal cannula and mask oxygen and may be used when the tidal volume of the patient is between 300 and 700 cc, the ventilatory rate is less than 30/min, and there is relatively stable ventilatory pattern with time. Because of the potential danger of excess oxygen administration, we prefer to keep the \text{FI}_{\text{O}_2} as low as is consistent with adequate oxygen delivery to the tissues. The unpredictability of the tidal volume and minute ventilation in an acutely ill patient are reasons why we generally recommend high-flow oxygen systems; the most commonly used of these are masks that work on the Venturi principle. The standard inspired \text{O}_2 fractions available are 24%, 28%, 35%, and 40%. Humidifiers are not required with these masks because the major portion of inspired gas is room air. To supply a high flow of oxygen in excess of 40%, special systems can be arranged by the Respiratory Therapy Department.

The importance of the mucociliary escalator in maintaining airway homeostasis is often underestimated. If the mucous blanket becomes dry or
abnormally thick, secretions will not mobilize normally and cough will become ineffective, leading to dramatic increases in airway resistance. The most effective means of reestablishing the mucous blanket is to hydrate the tracheobronchial tree. Water may be delivered to the airway in molecular form as humidification or in particulate form as an aerosol. Simple bubble-type humidifiers are sufficient for patients with an intact upper airway. Should the nasopharynx be bypassed by an artificial airway, a heater is added to the humidifier to further increase water content. If one wishes to deliver particulate water or medications to aid bronchial hygiene, a nebulizing device should be used. Ultrasonic nebulizers produce a very heavy water mist with uniform particle size on the order of .5µ to 3µ. Jet nebulizers, which operate on the Bernoulli principle, may be used for aerosolization of both water and medications. Droplets are propelled against baffles which eliminate the larger and heavier particles since droplets of greater than 10µ in size are clinically useless as they do not deposit in the tracheobronchial tree. Aerosolized medications include mucolytics, bronchodilators, vasoconstrictors, and steroids. In addition to instillation of water into the airway, efforts have been made to change the viscosity of the secretions by nebulizing sodium bicarbonate or acetylcysteine. Usually these agents are no better at mobilizing dried secretions than good hydration; however, in circumstances where the mucous is abnormal, such as cystic fibrosis, they may be useful. They may also be successfully used in those patients where active oral or intravenous hydration is not practical or safe. Inhaled bronchodilators exert their effect following absorption into the circulation. Absorption from the pulmonary system is rapid and the degree of bronchodilation obtained will be equal to that of an intravenous dose with similar blood levels. Vasoconstrictors may be administered by aerosol primarily for their topical properties. Many acute pulmonary diseases are accompanied by an inflammatory reaction of the respiratory mucosa which includes profound capillary congestion and swelling. Topical application of a vasoconstrictor may have dramatic effects in decreasing the hyperemia and increasing lumen size. Racemic epinephrine is a very effective topical vasoconstrictor and a mild systemic bronchodilator. It may be of significant benefit as adjunctive therapy to intravenous theophylline. Choice of nebulizing devices for any of these medications should be made by assessing the patient’s clinical status and the ability of the patient to generate adequate tidal volumes. We prefer that a hand-held nebulizer be used since these devices are inexpensive and are powered from a readily available compressed air source. There is evidence of improved distribution of particles when spontaneous inspiration is used with these nebulizers in contrast to medication delivered via intermittent positive pressure breathing (IPPB). These attempts at reducing airway resistance may be applied in conjunction with “expansion” therapy. Incentive spirometry appears to be superior to IPPB for preventing and treating atelectasis.

Despite the above measures (controlled oxygen administration, aerosolized medications, aggressive management of secretions, and expansion therapy) a number of patients with acute respiratory failure due to increased airway resistance will require intubation and mechanical ventilation. A volume-cycled ventilator is the modality of choice in acute respiratory failure. We follow the usual guidelines of 15 cc tidal volume/kg of body weight, respiratory rate of 15 times per minute and F O 2 of 40% for initial setting. Twenty minutes to a half hour after beginning mechanical ventilation, the arterial blood gases are repeated and adjustments are made as necessary. Because the basic problem with these patients is varying degrees of airway resistance in different areas of the lung, it is advisable to use as low an inspiratory flow rate as is consistent with the patient’s ventilatory pattern. This promotes gas delivery to poorly ventilated regions rather than over-expansion of well-ventilated regions. It is also important to provide for sufficient expiratory time. If too short a period is allowed for expiration, hyperinflation of the patient’s lungs because of gas trapping may result. A most useful recent addition to our therapeutic techniques is intermittent mandatory ventilation (IMV). A circuit of constant gas flow is added in parallel to the patient’s ventilator allowing the patient to breathe spontaneously at his or her own tidal volume between pre-set breaths delivered by the volume ventilator. This system minimizes the possibility of excessive mechanical ventilation and allows better acid-base control. As the patient’s clinical condition improves, the respirator rate may be gradually reduced, promoting a smoother weaning period. Complications that may develop in patients on ventilators include atelectasis in poorly ventilated areas, pneumothorax, superinfection, or drying of secretions with mucous plugging if the inspired atmosphere is not properly humidified.
Hypoxemic Hypocapnic Respiratory Failure

This blood gas pattern is found in syndromes in which the pulmonary parenchyma has been damaged. The severity of the condition will be apparent when there is a minimal increase in arterial oxygen tension in response to an $F_1O_2$ of .4 or above. This documents the presence of right-to-left shunting of blood. The reduction of parenchymal compliance in these patients makes the selection of tidal volumes more complex than in those with obstructive problems. The pressure volume curve of the respiratory system may be determined at the time the patient is placed on mechanical ventilation. We are often able to identify a point in the curve above which a slight increase in tidal volume produces a dramatic increase in intrathoracic pressure. This may occur at a tidal volume as low as .7 liters. If one arbitrarily applies the rule of 15 cc/kg to these patients, the thoracic capacity may be exceeded and there will be an inappropriately high intrathoracic pressure relative to the effective tidal volume. An appropriate minute ventilation may be delivered by reducing the tidal volume and increasing the rate of ventilation. This will reduce mean intrathoracic pressure and reduce the incidence of barotrauma.

Most patients in this category who are gravely ill will remain hypoxemic despite the increased $F_1O_2$ and mechanical ventilation. These modalities do not reverse the primary defect of reduced Functional Residual Capacity. This problem may be partially corrected by increasing the distending pressure within the thorax. This is done by applying positive end expiratory pressure (PEEP). Complications include a reduction in cardiac output and pneumothorax. It should be noted, however, that the occurrence of pneumothorax is related to the level of mean intrathoracic pressure during mechanical ventilation and not to the presence or absence of PEEP.

In summary, respiratory therapy should be thought of as a series of specific steps designed to treat the anatomic and physiologic alterations that occur with the various forms of respiratory failure. The scientific basis of the field is rapidly advancing as more is learned about the mechanisms of pulmonary disease. Respiratory therapy should no longer be equated solely with IPPB but should be regarded as a field encompassing a number of effective treatment modalities.

Suggestions for further reading


Screening Pulmonary Function Tests

GEORGE W. BURKE, III, M.D.

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The role of the Pulmonary Function Laboratory has been expanded in recent years by the commercial development and marketing of equipment capable of measuring accurately and easily static lung volumes, diffusing capacity, and arterial blood gases. These sophisticated measurements, which were once the purview of research physiologists, are now readily attainable as screening measurements in most community hospitals. This review is intended not as a summary of the entire field or as a technical guide for performance of pulmonary function tests but as a survey of some clinical applications and pitfalls of screening tests and a statement of guidelines for their use. It is assumed that the reader already has some experience in ordering and interpreting routine spirometric and arterial blood gases.

Pulmonary function tests are intended to answer four questions:
1) Is the patient restricted?
2) Is the patient obstructed?
3) Does the patient have abnormalities of gas exchange?
4) Are the observed abnormalities compatible with the patient's symptoms and clinical diagnosis?

Restriction is a reduction of total lung capacity or one of its subvolumes (Fig 1) as a result of collapse or volume displacement of the lung (fibrothorax), increased elastic recoil of the lungs (pulmonary fibrosis), increased elastic recoil of the rib cage or abdomen (obesity), or loss of forces needed to overcome normal elastic recoil (muscular dystrophy). It is important to realize that a routine spirometric vital capacity measurement may not adequately describe the presence or degree of restriction. For example, some patients with advanced emphysema may have reduced vital capacity in the face of a large residual volume, with increased total lung capacity caused by obstructive air trapping; it is misleading to label such patients as "restricted" on the basis of reduced vital capacity when, in fact, they are overinflated. On the other hand, true early restriction may occur in the face of normal vital capacity. An example of this is obesity, a condition in which the abdominal mass renders the chest wall less compliant by hindering downward displacement of the diaphragm. The earliest change in the restrictive pattern of obesity is a marked reduction in expiratory reserve volume (ERV) (see Fig 1) but only minor alterations of the total lung and vital capacities. Therefore, accurate evaluation of patients with suspected restrictive disorders should include, in addition to routine spirometry, a measurement of total lung capacity and its subdivisions by inert gas or plethysmographic techniques. The following case illustrates the use of pulmonary function studies in following the course of a restrictive lung disorder:

Case 1. A 53-year-old white woman developed exertional dyspnea and cough in 1971. A chest roentgenogram revealed bilateral basilar "ground-glass" opacities. In April 1972, an open lung biopsy showed desquamative interstitial pneumonia. She was placed on 20 mg prednisone daily in August 1972, and sequential pulmonary function tests were performed (Table 1). The initial vital and total lung capacities were consistent with moderately severe restriction, and there was a correspondingly severe reduction in diffusing capacity. After two years of prednisone therapy, these abnormalities improved substantially (October 1974). When steroids were stopped she experienced a severe relapse.
Obstructive impairment, defined as a reduction in inspiratory or expiratory air-flow rates, is a result of intrinsic airway disease or loss of lung elastic recoil. The latter causes airways to lose their structural support. Intrinsic airway disease may be caused by a variety of conditions ranging from totally reversible asthmatic bronchoconstriction to irreversible advanced chronic bronchitis with bronchiolitis oblitterans. Loss of elastic recoil almost always is a result of localized or generalized emphysema.

Because the conditions causing airways obstruction tend to become more exaggerated during forced expiration (during which alveoli and airways are rapidly diminishing in both length and diameter), the conventional method of assessing obstructive impairment is by measuring averaged flow rates on a volume-time curve of an ordinary spirogram during a maximally forced expiration of the vital capacity (Fig 2). Although one could arbitrarily measure flow at a multitude of points on the curve, three measurements have been standardized and used most frequently (see Fig 2): the timed vital capacity (FEV₁); the ratio of FEV₁ to total vital capacity; and the maximum mid-expiratory flow rate (MMEF) (the averaged flow over the mid-half of the vital capacity). Ordinarily all three measurements are made and abnormalities of one tend to correlate with those of the others. Additional measurements such as peak flow, total expiratory time, and forced expiratory volume in three seconds, or “FEV₃,” add little new information but may be sensitive indicators of changes when evaluating drug effects in population samples.

Obstruction is conventionally quantitated during expiration; however, patients develop dyspnea from obstructive impairment because of the increased work of breathing during inspiration. In most cases, obstruction during inspiration is inferred by abnormalities in the FEV₁ and MMEF; unfortunately, there are several important exceptions to this. Individuals with obstruction isolated to the trachea or larynx (for example, tracheomalacia following endotracheal intubation) may not have evidence of ex-

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Sequential Function Studies in a Patient with Desquamative Interstitial Pneumonia.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity, liters</td>
<td>2.06</td>
</tr>
<tr>
<td>Total lung capacity, liters</td>
<td>3.55</td>
</tr>
<tr>
<td>Arterial Po₂, torr</td>
<td>66</td>
</tr>
<tr>
<td>Diffusing capacity, ml/min/torr</td>
<td>8.5</td>
</tr>
</tbody>
</table>

(February 1976) which was reversed by their resumption (February 1977). In 1977 she was in remission while taking 20 mg prednisone on alternate days. The pulmonary function studies provided quantitative objective evidence that a prolonged course of steroid therapy was necessary.
spiratory obstruction if their defect is above the thoracic outlet, or they may have spirometric abnormalities which are misinterpreted as generalized airways obstruction if their defect is below the thoracic outlet. In addition, individuals with substantial narrowing of bronchioles but normal patency of bronchi (for example, young smokers with early chronic bronchitis) may have completely normal spirometric flow rates but isolated abnormalities of MMEF which are difficult to interpret because of the large standard deviation of this test in the normal population.

When either of these exceptions is suspected, additional information may be obtained by a recording of a flow-volume curve, or graphical representation of instantaneous mouth flow and expired volume during a forced inspiration and expiration of the vital capacity (Fig 3). The normal configuration of this curve is shown in Figure 3a. Fixed obstruction in the trachea produces a characteristic fixed limitation to flow which is independent of volume (Fig 3b). An additional advantage of the flow-volume curve is its depiction of inspiratory events, thus facilitating the detection of variable extrathoracic upper-airway obstruction (Fig 3c). Generalized bronchiolar narrowing produces flow limitations which are detectable only on expiration at lower lung volumes and tend to worsen as volume diminishes. This will result in an "inwardly concave" appearance to the flow-volume curve in patients with obstruction limited to the bronchioles (Fig 3d). It should be pointed out that this "inwardly concave" configuration is not specific for peripheral small airways obstruction; if the spirogram also indicates obstruction, the curve is not use-
ful for differentiating generalized obstruction from that isolated to peripheral airways. The following case is an example of obstructive impairment and also illustrates the value of lung volume determinations by the helium dilutional method in differentiating obstructive from restrictive impairment:

Case 2. A 19-year-old male gas station attendant abruptly developed exertional dyspnea in January 1976. Seven months later a carefully taken history revealed that he had been caring for his brother’s homing pigeons since late December 1975. The chest roentgenogram was normal, although symptoms persisted. Pulmonary function studies (Table 2, July 1976) revealed a reduced vital capacity; however, the total lung capacity and residual volume suggested probable obstructive air trapping rather than restriction. The FEV₁ and MMEF confirmed obstruction. Without specific treatment other than ceasing the pigeon exposure, his symptoms resolved and objective improvement was evident on sequential studies (September 1976). Although both restriction and obstruction may occur in allergic alveolitis, restriction is usually the dominant impairment. However, in this patient’s case, obstruction occurred without restriction.

Arterial hypoxemia is a sequela of impaired gas exchange; however, measurement of resting arterial oxygen tension (PₐO₂) alone may not be sufficiently sensitive to detect such impairment. Impaired gas exchange is also present if there is an increase in the observed oxygen tension difference between alveolar air and arterial blood (DA-a O₂) or if there is an abnormality in the diffusing capacity for carbon monoxide. For ambulatory patients breathing room air, DA-a O₂ can be estimated by obtaining a sample of arterial blood under steady-state conditions. Alveolar oxygen tension is calculated by the standard alveolar air equation:

\[ P_aO_2 = F_1O_2(713) = Paco_2 \left( F_1O_2 + \frac{1-F_1O_2}{RQ} \right) \]

\[ F_1O_2 = \text{fraction of } O_2 \text{ in inspired air} \]

\[ Paco_2 = \text{arterial } Pco_2 \text{ (identical to mean alveolar } Pco_2) \]

\[ RQ = \text{respiratory quotient} \]

The achievement of steady-state conditions is an essential prerequisite for this test because the estimation of alveolar oxygen tension is based on the assumption that the ratio of CO₂ produced to O₂ consumed (RQ) is 0.8. Transient hyperventilation caused by anxiety or pain will invalidate this assumption. Direct measurement of RQ may provide a more refined estimation of \( P_aO_2 \); however, this also requires sampling under steady-state conditions. For either approach, hyperventilation may be avoided by inserting an arterial cannula and allowing the patient to rest prior to sample collection.

The measurement of carbon monoxide diffusing capacity by the simplified single-breath technique requires the patient to breathhold at total lung capacity for ten seconds. Patients who are markedly dyspneic at rest may not be able to perform this test. Despite these limitations, useful results are forthcoming for most cooperative subjects.

The precise pathophysiologic explanation for impaired diffusion of carbon monoxide in lung diseases is unknown. Most authorities agree that it is a sign of alveolar or capillary destruction. Therefore, impaired CO uptake may occur in parenchymal destructive processes such as idiopathic fibrosing alveolitis, scleroderma, advanced sarcoidosis, and in vascular oblitative processes such as idiopathic pulmonary hypertension or multiple small pulmonary emboli. It is also impaired in processes causing reversible loss of functioning alveoli, examples of which are bacterial pneumonia and alveolar proteinosis. CO uptake is impaired in generalized emphysema due to emphysematous destruction of alveoli but remains nearly normal in chronic bronchitis because the destructive process is confined to airways and centrilobular elements of the parenchyma. Diffusing capacity may be spuriously impaired by several conditions. The first is the presence of severe anemia, which limits the availability of hemoglobin CO receptors; the second is that failure to achieve true total lung capacity during the breathhold will reduce CO uptake in proportion to the reduction in potential lung volume. The following case illustrates the value of the
TABLE 3

Pulmonary Function Studies in a Patient with Sarcoidosis

<table>
<thead>
<tr>
<th></th>
<th>September 1978</th>
<th>Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity, liters</td>
<td>2.98</td>
<td>3.63</td>
</tr>
<tr>
<td>FEV₁, liters</td>
<td>2.68</td>
<td>2.88</td>
</tr>
<tr>
<td>FEV₁/vital capacity, %</td>
<td>90</td>
<td>79</td>
</tr>
<tr>
<td>MMEF, liters/second</td>
<td>4.03</td>
<td>3.37</td>
</tr>
<tr>
<td>Total lung capacity, liters</td>
<td>3.85</td>
<td>5.12</td>
</tr>
<tr>
<td>Residual volume, liters</td>
<td>.76</td>
<td>1.49</td>
</tr>
<tr>
<td>Pao₂, torr</td>
<td>92</td>
<td>90</td>
</tr>
<tr>
<td>DA-a O₂, torr</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Diffusing capacity, ml/min/torr</td>
<td>12.0</td>
<td>21.7</td>
</tr>
</tbody>
</table>

Diffusing capacity in screening patients for the presence of gas exchange impairment:

Case 3. A 32-year-old woman complained of mild exertional dyspnea of two months duration. Chest roentgenogram revealed bilateral hilar adenopathy. Pulmonary function studies (Table 3) showed a modest reduction in total lung capacity (75% predicted) with normal vital capacity and flow rates. Resting arterial oxygen tension and DA-a O₂ were also normal. However, a moderately severe reduction in diffusing capacity suggested the presence of widespread interstitial disease despite the clear roentgenographic lung fields. Hemoglobin was normal. A transbronchoscopic lung biopsy showed interstitial non-caseating granulomata consistent with sarcoidosis.

Although measured impairment in pulmonary function frequently provides an objective explanation for the presence of dyspnea, many patients may experience this symptom because of abnormalities which are undetectable on screening evaluations. Negative screening studies should stimulate a careful search for vascular disease or nonpulmonary causes of dyspnea. For example, "metabolically justified" hyperpnea and dyspnea may accompany hyperthyroidism; patients with mitral stenosis or early congestive heart failure may have only mild impairment in lung mechanics despite severe exertional dyspnea; and patients with partial obliteration of the pulmonary vasculature may have normal lung mechanics, arterial oxygen tensions, and diffusing capacities while at rest, only to show abnormalities during exercise.

For a variety of vascular and interstitial pulmonary disorders, it may be appropriate to evaluate

TABLE 4

Guidelines for Ordering Medical or Surgical Screening Pulmonary Function Studies.

<table>
<thead>
<tr>
<th>1. Asymptomatic non-smoker</th>
<th>No studies; “routine spirogram” (vital capacity, FEV₁, MMEF) if thoracic surgery is planned.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Asymptomatic smoker</td>
<td>Routine spirogram; add resting arterial blood gases if thoracic surgery is planned.</td>
</tr>
<tr>
<td>3. Heavy smoker with dyspnea and/or chronic bronchitis</td>
<td>Routine spirogram; total lung capacity; diffusing capacity; resting arterial blood gases.</td>
</tr>
<tr>
<td>4. Young asthmatic</td>
<td>Spirogram before and after an inhaled bronchodilator.</td>
</tr>
<tr>
<td>5. “Old” asthmatic (&gt;30 years)</td>
<td>Spirogram before and after inhaled bronchodilator; total lung capacity; resting arterial blood gases.</td>
</tr>
<tr>
<td>6. Suspected tracheal obstruction, stridor, or unexplained obstruction on a previous spirogram</td>
<td>Inspiratory and expiratory flow-volume curve.</td>
</tr>
<tr>
<td>7. Suspected interstitial lung disease</td>
<td>Spirogram; total lung capacity; diffusing capacity; steady-state arterial blood gases with DA-a O₂.</td>
</tr>
<tr>
<td>8. Suspected pulmonary vascular disease</td>
<td>Same as #7. Add exercise DA-a O₂, physiologic deadspace, end tidal CO₂ gradient.</td>
</tr>
<tr>
<td>9. Suspected anatomic right-to-left shunt</td>
<td>Arterial blood gases while breathing 100% O₂. This study should never be requested for patients with emphysema or chronic bronchitis who have CO₂ retention.</td>
</tr>
<tr>
<td>10. Suspected early small-airways disease</td>
<td>In view of the limited clinical benefits, adequate evaluation is too expensive.</td>
</tr>
</tbody>
</table>
physiology during exercise as resting abnormalities frequently are exaggerated during exercise. However, pulmonary exercise testing, unlike cardiac stress testing, should provide a graded low level of exercise (doubling or tripling of the oxygen uptake) rather than maximum stress. In fact, recent experimental evidence has suggested that maximum stress measurements are potentially misleading because normal subjects may demonstrate transient abnormalities in $\text{Pao}_2$ and DA-a $\text{O}_2$ while performing temporary but severe tasks such as rapid stair-climbing. Although a complete discussion of exercise physiology testing is beyond the scope of this paper, some recent reviews of the relevance of pulmonary exercise testing in clinical medicine are included in the bibliography.¹,²

Table 4 is a suggested modus operandi for ordering screening pulmonary function studies for specific problems. Physicians ordering such tests must realize that measurements of lung mechanics require both cooperation and effort. Too much testing at a single sitting may fatigue the patient and cause spuriously abnormal results. For example, it would be unwise to subject a patient to both screening studies and exercise physiology studies for suspected vascular disease on the same day. Therefore, in the context of the suggestions in Table 4, clinical wisdom and common sense should dictate which tests are appropriate for individual patients.

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Flexible Fiberoptic Bronchoscopy

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The flexible fiberoptic bronchoscope was introduced in Japan by Dr. Shigeto Ikeda in the mid-1960s and became available for clinical use in the United States around 1970. The application of this technique represents one of the most significant advances for the diagnosis and management of chest diseases as it enables the physician to directly visualize the tracheobronchial tree and obtain diagnostic specimens from regions of the lung previously inaccessible to the rigid bronchoscope. Except for subpleural lesions, fiberoptic bronchoscopy is the surgical procedure of choice in the evaluation of many pulmonary lesions. In addition, fiberoptic bronchoscopy plays a major therapeutic role in the evaluation of airway patency and elimination of retained secretions.

The new Olympus® (BF-IT), Machida® (FBS-6TL; FBS-6TL II), and Pentax® (FB-19A) fiberoptic bronchoscopes have larger channels measuring 2.6 mm in diameter as compared with older models whose channels measure 1.8 to 2.2 mm. These larger channels provide more effective suctioning and larger biopsy specimens.

Indications for Fiberoptic Bronchoscopy

The following are accepted indications for fiberoptic bronchoscopy:

1. Any pulmonary lesion of uncertain nature. Subpleural lesions are exceptions.
2. Retained bronchial secretions
3. Evaluation of airway patency
4. Miscellaneous:
   a. hemoptysis in a patient with a normal chest radiograph
   b. positive sputum cytology (that is, cancer) in a patient with normal chest radiograph
   c. small, peripheral foreign bodies in adults
   d. patients with trauma or disease affecting the cervical spine or jaw
   e. patients on mechanical ventilators

Rigid bronchoscopy is preferred in the following situations:

1. Severe pulmonary hemorrhage
2. Centrally located foreign bodies
3. Children requiring bronchoscopy
4. Tracheal stenosis, secondary to intrinsic or extrinsic lesions

Risks and Contraindications

The following conditions are associated with increased risks during or following fiberoptic bronchoscopy:

1. Hemoptysis (increased incidence of post-bronchoscopy bleeding)
2. Bronchial asthma (danger of severe bronchospasm)
3. Uremia (danger of severe hemorrhage after biopsy)
4. Immunosuppression (hazard of post-bronchoscopy infection)
5. Superior vena cava obstruction (danger of inducing laryngeal edema)

Poor patient cooperation, hypoxemia with an arterial Po2 that cannot be increased to 60 to 65 torr with supplemental oxygen, uncorrected bleeding diathesis, acute respiratory acidosis of any degree, dangerous cardiac arrhythmias, recent (within six weeks) acute myocardial infarction, and untreated active pulmonary tuberculosis are contraindications for fiberoptic bronchoscopy. Terminally ill and aged patients with unstable vital signs should not have fibero-
optical bronchoscopy performed. Severe pulmonary hypertension and poor cardiopulmonary reserve are relative contraindications.

Physician and Patient Preparations for Fiberoptic Bronchoscopy

Prior to bronchoscopy, the endoscopist should perform a history and physical examination, review the chest radiographs, electrocardiograms, and blood and urine tests. Arterial blood gas and spirometric studies should be performed, if possible, prior to the procedure. Coagulation studies (prothrombin time, partial thromboplastin time, platelet count) should be obtained. Three sputum smears for acid-fast organisms should be obtained in patients whose chest radiographs are consistent with a diagnosis of tuberculosis. Finally, a signed operative permit should be obtained from the patient.

Oxygen prophylaxis. All patients should receive supplemental oxygen administered by nasal catheter or cannula 5 L/min. The \( P_{\text{a}O_2} \) may decrease as much as 20 torr during and for several hours after bronchoscopy.\(^4\) This drop in oxygen tension appears to be the result of multiple factors, including partial or complete obstruction of the airways, filling of alveolar spaces with lavage or anesthetic solutions, and suctioning. Therefore, all patients who have a reduced \( P_{\text{a}O_2} \) prior to the procedure should be given supplemental oxygen for six to ten hours following bronchoscopy.

Cardiac monitor. It is desirable that all patients should have cardiac monitoring during bronchoscopy. The sudden development of an arrhythmia or change in cardiac rate may suggest hypoxemia. Recent studies suggest that fiberoptic bronchoscopy does not consistently enhance preexisting ectopic beats in patients with ischemic heart disease, therefore, the procedure is not contraindicated in patients with stable angina. However, patients with coronary heart disease should be monitored closely during the procedure, since sinus tachycardia can develop, which may cause ischemic events thus precipitating arrhythmias.\(^8\) The most important factor in the development of arrhythmias is not bronchoscopy itself, but rather the underlying cardiopulmonary status of the patient.

Preoperative orders. For a morning procedure, NPO after midnight is required; for an afternoon procedure, only a light liquid breakfast may be given. Atropine 0.8 to 1.0 mg is given intramuscularly 30 minutes before bronchoscopy since this drug blocks vasovagal reflexes and also reduces the amount of bronchial secretions. Depending upon the age, size and clinical condition of the patient, morphine 7.5 to 15 mg or meperidine 50 to 100 mg may be given intramuscularly with the atropine. In our institution we commonly use codeine 60 mg and atropine 0.8 to 1.0 mg 30 minutes prior to bronchoscopy. Patients with severely reduced pulmonary function are given only atropine.

Topical anesthesia is an extremely important factor in securing patient acceptance and performing a successful bronchoscopic examination. Lidocaine is the most commonly used agent as it is effective and safe provided a maximum dosage of 600 mg is not exceeded during the procedure. This dose, which is larger than that usually recommended, has been found safe in a large series. In patients with severe liver disease or congestive heart failure no more than 300 mg should be given.

The patient is first instructed to gargle with approximately 4 ml of 4% lidocaine for one to two minutes, followed by spraying of the oropharynx and the most widely patent nostril with 5 ml of 4% lidocaine via a #15 DeVilbus atomizer. Cotton balls soaked in 4% lidocaine are applied to each piriform sinus for one minute; then, via a curved cannula, 2.0 ml of 1% lidocaine is administered over the vocal cords.

With the patient either seated or supine the flexible fiberoptic bronchoscope can be passed (with or without an endotracheal tube) either transnasally or transorally. The most commonly used technique for diagnostic study in the United States is the direct transnasal passage. (If fiberoptic bronchoscopy is carried out through an endotracheal tube, the internal diameter of the tube should be 8.5 mm). The distal six inches of the fiberoptic bronchoscope is lubricated with lidocaine jelly. The bronchoscope is then passed through the nostril and when in the oropharynx, the tip angulated anteriorly to view the larynx. If the patient begins coughing, 2 ml of 1% lidocaine is administered over the vocal cords. The patient is instructed to take a deep breath, and the bronchoscope is passed into the trachea during this inspiration. Additional small amounts of 1% lidocaine are given as necessary to suppress coughing. Once the bronchoscope is in the tracheobronchial tree, examination of all five lobes can be performed in approximately 10 to 15 minutes.

Biopsy Technique

A variety of biopsy instruments, primarily brushes and forceps, are available for diagnosis of
pulmonary lesions. In general, both brushes and forceps are used for all lesions. The brush biopsy is obtained by moving the brush briskly back and forth over the suspicious area. If the bronchoscope has been introduced through the endotracheal tube, after each biopsy, the brush is pulled back just inside the bronchoscope and the bronchoscope removed from the tracheobronchial tree. The brush is then advanced forward and smears are made on glass slides which are placed in 95% alcohol.

Forceps biopsy can be used in the diagnosis of local or diffuse pulmonary lesions. In diffuse lung disease, the forceps is introduced into a small peripheral airway, usually in a lower lobe segment. The forceps is opened in inspiration, the patient is instructed to exhale completely, the forceps is advanced slowly about 1 cm, and at the end of an exhalation the biopsy obtained. Following the removal of the forceps, the distal end of the bronchoscope should be wedged into the bronchial segment for one minute to prevent possible bleeding. The biopsy specimen is placed in a sterile tube filled with Ringer solution. In general, three to four specimens, sometimes as many as six to eight, are taken. Bronchial washings with 10 to 20 ml saline solution should also be obtained. The specimens should be sent for cytology, smears, and cultures for acid-fast organisms, fungi, and pathogenic organisms.

It is desirable to perform transbronchial biopsy under fluoroscopic control if available. Because of the possibility of bilateral pneumothorax, biopsies are taken from one lung only.

In localized disease the forceps is introduced to the lung lesion, then withdrawn 1 cm, opened, then advanced until it touches the lesion; the forceps is closed and the biopsy obtained. Prior to brush or forceps biopsy, administration of 5 ml boluses of 1:20,000 epinephrine over visible tumors or into distal airways (in cases of diffuse lesions) before biopsy, reduces the incidence of pulmonary hemorrhage. A total of 20 ml of 1:20,000 epinephrine solution can be given over a period of 20 minutes, except in patients with severe hypertension and serious arrhythmias. In patients with uremia, brush or forceps biopsies should not be done because of the possibility of severe hemorrhage; in those with a platelet count of less than 50,000/mm³, six to ten platelet packs should be infused just prior to bronchoscopy. Finally, patients with bronchial asthma are prone to develop severe bronchospasm. They should be given bronchodilators and probably steroids prior to bronchoscopy.

Where centrally located visible carcinoma of the lung is present, the incidence of positive forceps biopsy is about 97%. The overall yield in peripheral carcinoma of the lung is about 60% to 70%, in metastatic carcinoma of the lung, about 30% to 40%, and in diffuse lung diseases, the diagnostic yield ranges from 62% to 79%. In stage I sarcoidosis, transbronchial biopsy will confirm the diagnosis in 50% to 60% of cases while in stage II and stage III sarcoidosis, diagnostic success is about 90%.

Complications of Fiberoptic Bronchoscopy

The overall complication rate is approximately 8%. The mortality rate is 0.1%. The following are potential complications:

1. Hypoxemia
2. Laryngospasm
3. Hemorrhage
4. Pneumothorax
5. Bronchospasm
6. Cardiac arrhythmias and acute myocardial infarction
7. Post-bronchoscopy infection
8. Trauma due to endotracheal tube insertion
9. Hypoventilation

In summary, fiberoptic bronchoscopy is an extremely useful technique in the diagnosis and management of pulmonary diseases. It is quite safe and comfortable for the patient and it permits examination of airways previously inaccessible to the endoscopist. However, physicians must evaluate each patient carefully. The procedure should be performed by an experienced endoscopist who is familiar with risk factors and proper indications for fiberoptic bronchoscopy.

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Bedside Flow-Directed Balloon Catheterization in the Critically Ill Patient

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Prior to 1970, catheterization of the right heart and pulmonary artery required the use of fluoroscopic guidance and was only performed in specialized research units and/or the cardiac catheterization laboratory. The need to assess the hemodynamic status of the left atrium and ventricle on a continuing basis brought about the development of the flow-directed balloon-tipped catheter. With the availability of this tool, routine bedside right heart catheterization has become a reality in the critical care units of many community hospitals. This technique provides the physician with the means for indirectly appraising left heart hemodynamics and to gauge the effects of various modes of therapy on tissue perfusion by monitoring the pulmonary artery and pulmonary capillary wedge pressures (PCWP), cardiac outputs, and mixed venous PO₂ (PvO₂).

Relationship Between Pulmonary Capillary Wedge Pressure (PCWP) and Left Ventricular End Diastolic Pressure

The term pulmonary capillary wedge pressure originated many years before the discovery of the flow-directed catheter. During routine diagnostic right heart catheterizations, it was customary to advance the rigid catheter, under fluoroscopic guidance, from a large pulmonary artery to a more peripheral position. Here resistance was met within a small pulmonary artery branch whose inside diameter approximated that of the catheter. The catheter was then forcibly advanced a short distance until it became physically wedged. This “wedging” occluded the pulmonary artery pressure tracing and monitored the distal or downstream pressure, that is, left heart pressures were reflected retrograde through the pulmonary capillaries to the catheter tip. These same pressures can now be monitored at the bedside, employing the flow-directed catheter by inflating the balloon and temporarily obstructing pulmonary artery pressures and flow.

Since the PCWP is an indirect reflection of the left atrial pressure, it is used as an assessment of the left ventricular filling pressure during diastole. The value of this measurement is important in the diagnosis and management of the critically ill patient.

Indications for Bedside Pulmonary Artery Catheterization

Although there are no universally agreed upon, absolute indications for the use of the flow-directed catheter, the following list includes many of the clinical conditions in which hemodynamic monitoring may be useful.
1. As a guide to fluid management
   a. acute renal failure
   b. acute pancreatitis
   c. burn patients
2. Evaluation and management of shock states
   a. septic
   b. hypovolemic
   c. cardiogenic
3. Diagnosis and management of respiratory failure
   a. adult respiratory distress syndrome
   b. primary pulmonary hypertension
   c. pulmonary embolism
4. Diagnosis of cardiac dysfunction
   a. cardiac tamponade
   b. constrictive pericarditis
   c. mitral insufficiency
   d. intracardiac shunts (left to right)
5. Assessment of left ventricular function
   a. acute myocardial infarction
   b. congestive heart failure
   c. post-open heart surgery
   d. ventricular afterload reduction

Catheter Insertion

Our choice of insertion routes are the median basilic, internal jugular, femoral, and subclavian veins, in that order. (We are particularly hesitant to employ the subclavian approach because of the chance of pneumothorax which may be poorly tolerated in the critically ill patient). Percutaneous guide wire insertion (Fig. 1) is normally used, although on rare occasions a surgical cutdown of a median basilic vein may be necessary.

Following introduction, the catheter tip is advanced to the intrathoracic vena cava (a distance of 35 to 40 cm from the femoral or basilic vein and 15 to 20 cm from the internal jugular insertion site) where the balloon is then inflated with from 0.5 to 1 cc of air. With the balloon acting as a sail, the catheter is pushed through the right atrium and ventricle and into the pulmonary artery (Fig. 2). The position of the catheter tip is determined by the pressure contour tracing monitored throughout the procedure (Fig. 3). As the catheter tip reaches smaller caliber pulmonary arteries, the balloon occludes the vessel, producing a wedge pressure tracing. During catheter advancement, the electrocardiogram is carefully monitored by the nurse assisting with the procedure. The nurse reports the number of successive PVCs as a warning to the physician to change the position of the catheter tip.

Hemodynamic and Physiological Indices Derived from Flow-directed Catheters

**Pulmonary capillary wedge pressure (PCWP).** The PCWP reflects left ventricular end diastolic filling pressures in the absence of left atrial or mitral valve dysfunction (mitral stenosis, mitral insufficiency, left atrial myxoma, and other conditions). The normal pulmonary wedge pressure ranges from 6 to 12 mm Hg with a value greater than 12 mm Hg, implying either left ventricular dysfunction or fluid overload. To differentiate these two clinical conditions, a cardiac output and/or P\textsubscript{a}O\textsubscript{2} needs to be obtained. (See below) A low pulmonary wedge pressure, that is, below 6 mm Hg, implies "pure" intravascular fluid depletion such as occurs with hemorrhage, or...
“relative” intravascular depletion as is seen in the early stages of septic shock. Once again cardiac output or P\textsubscript{V\textsubscript{o\textsubscript{2}}} determinations will help differentiate these entities. (See below)

**Cardiac output.** Balloon-tipped flow-directed catheters are available with thermodilution cardiac output capabilities. These triple lumen catheters have an orifice located 30 cm proximal from the tip through which room temperature or iced saline, or 5% dextrose solutions, can be injected for the purpose of obtaining a cardiac output.\(^2\) A thermistor bead located near the tip of the catheter detects changes in temperature from which cardiac outputs can be computed.

**Mixed venous P\textsubscript{o\textsubscript{2}} (P\textsubscript{V\textsubscript{o\textsubscript{2}}}).** The flow-directed catheter also permits sampling of true mixed venous blood (obtained from the pulmonary artery) for P\textsubscript{o\textsubscript{2}} and oxygen content determinations. The P\textsubscript{V\textsubscript{o\textsubscript{2}}} (normal range in a nonanemic patient, 35 to 40 torr) reflects changes in cardiac output and tissue perfusion. As tissue perfusion and/or cardiac output decreases, the P\textsubscript{V\textsubscript{o\textsubscript{2}}} decreases as peripheral tissues extract more oxygen from the blood. For example, a patient with a high PCWP and a normal or elevated P\textsubscript{V\textsubscript{o\textsubscript{2}}} has a normal or elevated cardiac output thus implicating fluid overload as the cause for the elevated PCWP. In a patient whose PCWP is elevated and P\textsubscript{V\textsubscript{o\textsubscript{2}}} (and thus cardiac output) is low, tissue perfusion has been compromised due to left ventricular dysfunction. In contrast, a patient with a low PCWP and a low mixed P\textsubscript{V\textsubscript{o\textsubscript{2}}} (usually below 30 torr) probably has “pure” intravascular fluid depletion and will need intravenous fluids to correct the deficiency. A low PCWP and an inappropriately elevated P\textsubscript{V\textsubscript{o\textsubscript{2}}} (usually above 30 torr) is usually a sign of sepsis. In this condition peripheral arteriovenous shunting occurs with failure to extract oxygen at the cellular level.

**Shunt equation (Q\textsubscript{s}/Q\textsubscript{t}).** The degree of physiologic and/or anatomic shunting which occurs in patients with both cardiac and pulmonary disease(s) can be calculated by employing the standard shunt formula and obtaining true mixed venous oxygen contents from the flow-directed catheter when in the pulmonary artery position. The shunt calculation, although somewhat cumbersome and complicated, can be used as an index for weaning patients from ventilators, for determining the most appropriate level of positive end expiratory pressure (PEEP), and for following the improvement or deterioration in the patient’s cardiopulmonary status on a day-to-day basis.

**Complications**

As with any other invasion procedure, flow-directed catheterization can be associated with complications but probably in less than 1% to 2% of all insertions.\(^3\)

1. Ventricular and atrial arrhythmias, including ventricular tachycardia and fibrillation.
2. Pulmonary infarction
3. Ruptured cordae tendenae of the tricuspid valve.
4. Rupture of a pulmonary artery and/or capillary with subsequent hemorrhage
5. Infections

**The Nurse’s Role**

The critical care nurse plays an important and essential role during and following the catheterization. It is his or her responsibility not only to set up and calibrate the transducers but also to assist during the actual catheterization. The nurse must be proficient in monitoring the electrocardiogram during catheter placement and inform the physician of any potentially serious arrhythmias. Additionally, he or she must be an expert in monitoring and reporting
the pulmonary artery and pulmonary wedge pressures. Finally, the nurse must be acquainted with the problems associated with obtaining accurate pressure measurements and be alert to possible complications that can occur with the flow-directed catheter.

In summary, with the aid of the balloon-tipped flow-directed catheter, we are now able to measure pulmonary artery and pulmonary capillary wedge pressures at the bedside. The results obtained from pulmonary artery catheterization have added greatly to the management of the critical ill patient.

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Intrapulmonary Lymph Node Presenting as a ‘Coin’ Lesion: A Case Report

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Intrapulmonary lymph node seldom presents as a solitary pulmonary nodule. This rare clinical entity is reported in the following case:

Case Report

A 40-year-old woman was evaluated because of the radiological finding of a noncalcified, noncavitary, solitary, pulmonary nodule. She was asymptomatic at the time of examination. The patient's past history included hiatal hernia, duodenal ulcer, and recurrent supraventricular tachycardia. She has habitually smoked one pack of cigarettes daily for 15 years. The physical examination was unremarkable except for mild obesity.

A round density in the right infrapulmonary area was found in chest roentgenogram, which was not present in previous films. Tomographic examination showed a smooth mass without cavitation and calcification (Figure).

Bronchoscopic examination was normal and the patient underwent exploratory right thoracotomy. A firm, smooth mass was located in the anteromedial segment of the right lower lobe. A vertical incision was made directly over the lesion to a depth of approximately 2 cm through the lung tissue. The exposed nodule was completely excised and reported as benign intrapulmonary lymph node. The postoperative course was uneventful.

Discussion

An intrapulmonary lymph node large enough to present as a “coin” lesion on chest x-ray is rare. Steele reported only four such cases in 887 resected solitary pulmonary nodules. Lymph nodes are normally found around the main bronchi and its lobar branches, usually extending only as far as the third or fourth divisions. Only aggregations of lymphoid tissue are found beyond this point. According to Trapnell, Miller believed that the intrapulmonary lymph nodes developed in abnormal lung from lymphoid tissue. The lymphoid tissue in lungs would increase with age, probably in response to inhaled carbon particles or infections. Trapnell in his study of 92 postmortem lungs found six cases of intrapulmonary lymph node and concluded that “such nodes are not nearly so uncommon as is suggested by the literature.” Greenberg reported a case of subpleural lymph node in 1961. This was a hyperplastic lymph node caused by anthracotic particles. A similar case has been reported by Rosenthal and his associates.

Exploratory thoracotomy followed by resection continues to be the most commonly used method in the diagnosis and treatment of a solitary pulmonary nodule.

Summary

A case of intrapulmonary lymph node presenting as a solitary pulmonary nodule is presented with a brief discussion.
Acknowledgment: We thank Connie Powell for her assistance in the preparation of this manuscript.

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Maxillary Periapical Actinomycosis: A Case with an Unusual Roentgenographic Appearance

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Actinomycosis was once a fairly common disease and one that has a long history. “It was undoubtedly observed early in the 19th Century, as actinomycotic lesions were described erroneously in 1826 by LeBlanc as osteosarcomas and later in the 1800s Bollinger (1876) first recognized it as a specific entity which he named ‘lumpy jaw.’” The most frequent clinical form of actinomycosis is the cervicofacial type which is seen in 60% of reported cases, the other forms being abdominal (20%), pulmonary (15%), and cutaneous (5%). Young adult males are most frequently affected with actinomycosis. The actinomycetes, the so-called “higher” bacteria, are among the more common microorganisms found within the oral cavity; however, they seldom exist as pathogens within the oral cavity, and Goldstein et al (1972) report that there are fewer than 50 cases of actinomycosis of the maxilla reported in the English literature. Periapical actinomycosis is seen even less frequently. Browne and O’Riordan reported a case of periapical actinomycotic granuloma and found that only ten such cases were on record before 1966. In 1975 Samanta et al reported that the analysis of cases reported subsequently to those of Browne and O’Riordan revealed only five additional cases in which colonies of actinomycetes were demonstrated on histologic studies of the periapical tissue.

The diagnosis of actinomycosis may be accomplished by several means. While a direct smear of the pus and identification of the sulfur granules are suggestive of actinomycosis, anaerobic culture or histologic evidence, or both, are considered diagnostic. The roentgenographic appearance of cervicofacial actinomycosis is not diagnostic, although chronicity and a relative lack of bone reaction are suggestive. The appearance may vary from one of lytic destruction without bone formation to one of a definite thickening and sclerosis. The most common appearance of maxillary actinomycosis is a localized radiolucent periapical or periodontal abscess in a healthy adult who shows no signs of systemic toxicity. In distinction to mandibular actinomycosis, cutaneous fistulas or hard facial swellings are unusual in the maxillary form of the disease. Antral-facial fistulas as well as oral-antral fistulas have been noted from maxillary molar extraction sites. Intraoral mucosal drainage occurs much more frequently with maxillary actinomycosis than with the mandibular form of the disease. Oral trauma or a preexisting condition is a common feature of maxillary actinomycosis.

This paper describes a case of the rare form of maxillary periapical actinomycosis with a bizarre roentgenographic appearance. The clinical signs and symptoms along with the roentgenographic appearance of this lesion were not suggestive of acti-
Actinomycosis and thus were not included within the clinical differential diagnosis.

**Case Report**

A 33-year-old white female consulted her private practitioner, and upon radiographic examination, a radiolucent lesion associated with the apex of the upper left first molar was discovered (Fig 1). Questioning revealed that the lesion had been present for over three years during which time the patient had not sought treatment. Clinically, friable brownish tissue with multiple root fragments was surgically removed by excisional biopsy and sent to the laboratory for histologic examination.

Histologic examination revealed a multi-

- sectioned soft tissue mass composed of granulomatous fibrous connective tissue containing areas of central abscess formation from which radiating hyphae-like structures showing the characteristic eosinophilic “clubs” were seen (Fig 2). These “granules” appeared to be floating in a sea of polymorphonuclear leukocytes associated with an occasional macrophage and multinucleated giant cell (Fig 3). On the surface of this lesion several organisms having budding yeast-like forms scattered along pseudo-hyphae were noted (Fig 4). Pathologic diagnosis based on histologic examination with special stains was a mixed infection of actinomycosis and a superficial candidosis.

Following surgical removal of the lesion and
histologic diagnosis of the tissue, the patient's local practitioner began treatment with Penicillin V-K, 250 mg tablets four times per day for twenty days and nystatin (Mycostatin) (500,000 units), 1 tablet three times per day. Systemic involvement of the disease was ruled out by medical examination. One year following treatment, roentgenographs of the area showed complete resolution of the lesion (Fig 5).

Discussion

The unique feature of this case is the bizarre radiographic appearance of the lesion. In contrast to the normal localized radiolucent periapical lesion of maxillary periapical actinomycosis, this lesion appeared radiographically as a multilocular lesion ("soap bubble" appearance) more characteristic in this region of a giant cell granuloma, myxoma, or brown tumor of hyperparathyroidism.

Clinicians often associate actinomycosis with the more florid soft-tissue cervicofacial type and this is obviously not always found owing to the localization of maxillary lesions which tend to remain asymptomatic.

In terms of treatment in maxillary periapical lesions, Stenhouse\(^3\) notes that there appears to be no need for recourse to prolonged antibiotic therapy whenever surgical intervention alone can totally eradicate the infected focus. This case substantiates this claim.

Summary

An unusual case of maxillary periapical actinomycosis presenting with a bizarre roentgenographic appearance is presented. Local surgical eradication coupled with short-term drug therapy resulted in the complete resolution of the lesion.

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The Dental Health Status of Pre-Columbian Peruvians: A Study of Dental Caries, Missing Teeth, Attrition, Osteitis, Calculus, and Bone Loss

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Prior to the recent reports of Sawyer et al1 and Elzay et al2 on the characteristics and dental health status of ancient Peruvian cultures, only Stewart4, Leigh4, and Goaz and Miller5 had reported on the dental morphology and pathology of the pre-Columbian Peruvian Indians. The purpose of this study is to evaluate the dental health of these ancient peoples to further our understanding of the development of dental diseases. This paper follows up and expands the report of Elzay et al2 to include another culture and completely new specimens, with a look at primary dentitions not previously available for study.

Materials and Methods

The permanent maxillae from 115 individuals, and 113 permanent mandibles along with 46 individual primary maxillae and their primary mandibles, separated by cultural groups, were included in this study. Only four specimens were found which had a mixed dentition and these were not included in this study. Thus, those maxillae and mandibles labeled permanent had only permanent dentitions and those labeled primary had only primary dentitions. All examinations were completed by gross examination of materials with dental probes and explorers under op-
timal light. A dental radiographic unit was available and x-rays were used to confirm diagnoses.

The only teeth counted as missing were those apparently lost prior to death as shown by osseous healing in the edentulous area. Third molars were considered congenitally absent if there was no evidence of previous tooth loss and subsequent repair, or if they did not show on x-ray or were not grossly present. If the third molar crown appeared within the bone but had not completely erupted, the tooth was classified as either semi- or unerupted.

Attrition or abrasion with no gross loss of crown enamel was scored as 0; the tooth was scored as 1 if enamel loss on the cusp tips did not involve the grooves and fissures, as 2 if the enamel loss involved the grooves and fissures, and as 3 if the process eliminated the grooves and fissures on the occlusal surface. This system differs from the Attrition Index of Lavelle\textsuperscript{a} only in omitting a fourth category for teeth having pulp exposure from attrition and in recording the highest score (most severe) noted on the specimen in lieu of individual scores for all teeth of the specimen.

The incidence of dental caries was recorded in two ways. First, caries were recorded according to the DMFS (decayed, missing, filled surfaces) index. If a tooth had a carious lesion involving only one of its five surfaces, that is, mesial, distal, buccal, lingual and occlusal (incisal), it was scored as 1. A missing tooth (antemortem) or one with all five surfaces carious received a score of 5. This system denotes severity of disease whereas reporting the percent of teeth affected reports only the caries rate. The second method of recording specimens was a modification of the DMFS index. While most teeth today are lost as a result of dental caries, teeth may have been lost in ancient times through trauma, periodontal disease, or pulpitis from severe attrition. Hardwick\textsuperscript{7} developed a method, attempting to correct this; however, it is more meaningful to express caries incidence by omitting missing teeth. Hence, dental caries involvement is expressed as DS (decayed tooth surfaces) per tooth and jaw (specimen).

The condition of osteitis was recorded as present or absent only. Gross evidence of either bone destruction and/or proliferation with or without a fistulous tract was recorded as osteitis.

The degree of calculus formation was indicated as light if less than 1 mm of calculus was deposited on the lingual surfaces of the anterior teeth, moderate if concomitant deposition around posterior teeth was less than 1 mm, and heavy if calculus deposition on anterior or posterior teeth exceeded 1 mm.

Bone loss was recorded on a scale of 0 for no gross evidence, 1 for interdental bone loss between adjacent teeth, 2 for bone loss down to the bifurcation area of the roots, and 3 for bone loss beyond the bifurcation area. In any one jaw specimen only the most severe manifestation of bone loss was recorded.

Results

The results for the permanent dentitions and jaws are tabulated in Tables 1, 2 and 3, while the results for the primary dentitions and jaws are tabulated in Tables 4, 5 and 6. Individuals from the Ica culture and the Paracas culture (Table 1) exhibited the highest incidence of missing teeth in the maxillary arch, those from the Colonial culture showed the highest incidence of missing teeth in the mandible. The Nazca culture Indians followed by those of the Huari culture showed the fewest missing teeth in both arches. The Ica and Paracas culture Indians showed the highest incidence of missing first, second, and third molars in the maxillary arch while the Paracas and Colonial culture Indians had the highest incidence of missing first, second, and third mandibular molars. Individuals from the Paracas culture followed by those from the Colonial culture showed the highest incidence of congenitally missing third molars in both arches.

The Paracas, Ica and Colonial Indians had the highest incidence of dental caries in both arches in the permanent dentitions (Table 2). Nazca culture Indians showed a low incidence of dental caries while the Incas showed the lowest of all. In individuals with the permanent dentitions, those from the Nazca and Inca cultures had the highest incidence of osteitis in the two arches. The Paracas, Ica and Colonial Indians had the highest degree of moderate and heavy calculus. While there is little difference in the degree of bone loss in the six cultures, the Paracas Indians showed a somewhat higher degree of loss.

With regard to primary arches and dentitions, the only antemortem tooth losses were one tooth in the maxillae in the Colonial individuals and two teeth lost from the mandibular arches of the those from the Ica culture (Table 4). No first or second molars of either arch were lost in these specimens and there were no congenitally missing teeth. As with the permanent dentitions, the Paracas, Ica and Colonial Indians had the highest incidence of dental caries in both arches (Table 5). Individuals from these three
TABLE 1
Frequency of Missing or Unerupted Teeth in the Permanent Dentitions

<table>
<thead>
<tr>
<th>Culture</th>
<th>N</th>
<th>NTP</th>
<th>Teeth Lost</th>
<th>X Teeth Lost per JAW</th>
<th>% Missing</th>
<th>% Congenitally Missing</th>
<th>% With semi-or UNERUPTED</th>
<th>% Lacking</th>
<th>% Lacking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paracas</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>46</td>
<td>736</td>
<td>101</td>
<td>13.7</td>
<td>2.2</td>
<td>18.5</td>
<td>5.5</td>
<td>17.4</td>
<td>19.6</td>
</tr>
<tr>
<td>mandibular</td>
<td>31</td>
<td>496</td>
<td>64</td>
<td>12.9</td>
<td>2.1</td>
<td>19.4</td>
<td>4.9</td>
<td>24.2</td>
<td>35.5</td>
</tr>
<tr>
<td>Nazca</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>9</td>
<td>144</td>
<td>10</td>
<td>6.9</td>
<td>1.1</td>
<td>11.1</td>
<td>0.0</td>
<td>22.2</td>
<td>11.1</td>
</tr>
<tr>
<td>mandibular</td>
<td>20</td>
<td>320</td>
<td>30</td>
<td>9.4</td>
<td>1.5</td>
<td>12.5</td>
<td>0.0</td>
<td>10.0</td>
<td>16.7</td>
</tr>
<tr>
<td>Huari</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>13</td>
<td>208</td>
<td>19</td>
<td>9.1</td>
<td>1.5</td>
<td>11.5</td>
<td>0.0</td>
<td>23.1</td>
<td>19.2</td>
</tr>
<tr>
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<td>14</td>
<td>224</td>
<td>23</td>
<td>10.3</td>
<td>1.6</td>
<td>7.1</td>
<td>0.0</td>
<td>3.6</td>
<td>28.6</td>
</tr>
<tr>
<td>Ica</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>24</td>
<td>384</td>
<td>56</td>
<td>14.6</td>
<td>2.3</td>
<td>18.8</td>
<td>2.1</td>
<td>27.1</td>
<td>20.8</td>
</tr>
<tr>
<td>mandibular</td>
<td>25</td>
<td>400</td>
<td>52</td>
<td>13.0</td>
<td>2.1</td>
<td>16.0</td>
<td>4.0</td>
<td>24.0</td>
<td>34.0</td>
</tr>
<tr>
<td>Colonial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>20</td>
<td>320</td>
<td>42</td>
<td>13.1</td>
<td>2.1</td>
<td>12.5</td>
<td>0.0</td>
<td>57.7</td>
<td>2.5</td>
</tr>
<tr>
<td>mandibular</td>
<td>19</td>
<td>304</td>
<td>42</td>
<td>13.8</td>
<td>2.2</td>
<td>10.5</td>
<td>0.0</td>
<td>47.4</td>
<td>5.3</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>22</td>
<td>352</td>
<td>43.5</td>
<td>11.28</td>
<td>1.82</td>
<td>44.0</td>
<td>13.73</td>
<td>2.10</td>
<td>25.38</td>
</tr>
<tr>
<td>mandibular</td>
<td>21</td>
<td>346</td>
<td>44.3</td>
<td>12.63</td>
<td>2.02</td>
<td>43.3</td>
<td>14.08</td>
<td>2.28</td>
<td>19.38</td>
</tr>
</tbody>
</table>

N = number of jaw specimens
NTP = number of teeth possible
X = mean

Discussion

The results indicate that in the permanent dentitions the Paracas, Ica and Colonial individuals have the highest incidence of missing antemortem teeth while in the primary dentitions only the Ica and Colonial cultures have specimens showing tooth loss. The Nazca and Huari permanent dentitions exhibit the lowest incidence of antemortem tooth loss. These findings parallel dental caries incidence, as the Paracas, Ica and Colonial Indians have the highest incidence of dental caries in both the permanent and primary dentitions. The incidence of antemortem tooth loss has been reported to decline spectacularly with the transition to an agricultural society, from 41.6% loss in early hunting and gathering economies to 6.2% in the most recent phases. These pre-Columbian cultures were primarily agricultural societies. Average tooth loss in the permanent dentitions was 11.3% in the maxillary arch and 12.6% in the mandibular arch. In our earlier study consisting of 101 mandibles, an 18.6% antemortem loss was found. The tabulated findings on antemortem tooth loss in ten prehistoric adult populations show a 19.9% loss for Gran Canaria. The Inca individuals showed the highest incidence of semi- or unerupted third molars. This group also had low scores for caries, bone loss, and calculus, suggesting that although the sample was an adult population, it was a younger age group than the other cultures. (This was later confirmed by age analysis.)

The Paracas, Colonial and Ica Indians showed evidence of congenitally missing third molars. The Paracas individuals showed the highest incidence of congenitally missing third molars in both arches,
being 5.5% in the maxillae and 4.9% in the mandible. The Nazca, Huari and Inca Indians showed no specimen with congenitally missing teeth among those studied. The average for all six cultures was 2.1% for the maxillary third molars and 2.3% for the mandibular third molars.

While at least one of the cultures consisted of young adults, the incidence of third molar hypodontia was 15.8% (including those that were congenitally missing) for the maxillary arches and 16.4% (including the congenitally missing) for the mandibular arches. Carbonell reported a 2.6% incidence of third molar hypodontia (mandibles only) in the Kish of Mesopotamia (3000 B.C.) while Crispim et al reported 8% per quadrant and 2% for all four third molars in a trihybrid Brazilian population. Niswander reported a 30.5% incidence of one or more missing third molars in the Xavante Indians of Brazil, and Dahlberg indicated that Mongoloid people have a higher percentage of agenesis of third molars than do other groups. Our earlier data showed a third molar hypodontia of 3.6% in 101 mandibles. The present data more clearly confirms what one would expect in a population of Mongoloid background. An obvious difficulty in comparison of available data is the lack of uniformity in reporting results.

Our findings on attrition indicate that the six pre-Columbian cultures had moderate attrition and no resulting pulp exposures. Occlusal wear pattern was not noticeably oblique as noted in cultures where teeth are used to strip husk or bark from a food source. It would appear that the cultures shared a similar diet in terms of consistency and/or preparation. Previous reports on pre-Columbian Peruvians also found no severe attrition or pulp involvement. Similarly, Carbonell found no pulp involvement from attrition in ancient Mesopotamians. Although the data on partially erupted third molars suggest that the Inca specimens are of a younger age, these individuals still had attrition effects similar to the specimens of the other cultures.

Results of dental caries have meaning only when comparisons are made among the cultures observed. While lower age may cause the low caries in the Inca

---

**TABLE 2**

<table>
<thead>
<tr>
<th>CULTURE</th>
<th>N</th>
<th>X SCORE OF ATTENTION 0, 1, 2, 3</th>
<th>TNT</th>
<th>X DMFS/TOOTH X DMFS/JAW</th>
<th>X DS/TOOTH</th>
<th>X DS/JAW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paracas</td>
<td>45</td>
<td>1.8</td>
<td>434</td>
<td>2.23</td>
<td>19.20</td>
<td>1.08</td>
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<tr>
<td>maxillary</td>
<td>9</td>
<td>2.1</td>
<td>102</td>
<td>1.02</td>
<td>11.56</td>
<td>0.43</td>
</tr>
<tr>
<td>mandibular</td>
<td>20</td>
<td>2.2</td>
<td>264</td>
<td>1.03</td>
<td>13.65</td>
<td>0.47</td>
</tr>
<tr>
<td>Nazca</td>
<td>9</td>
<td>2.1</td>
<td>102</td>
<td>1.02</td>
<td>11.56</td>
<td>0.43</td>
</tr>
<tr>
<td>maxillary</td>
<td>13</td>
<td>1.6</td>
<td>142</td>
<td>1.37</td>
<td>15.00</td>
<td>0.70</td>
</tr>
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<td>mandibular</td>
<td>14</td>
<td>1.9</td>
<td>174</td>
<td>1.25</td>
<td>10.79</td>
<td>0.59</td>
</tr>
<tr>
<td>Huari</td>
<td>24</td>
<td>1.9</td>
<td>294</td>
<td>1.96</td>
<td>24.04</td>
<td>1.01</td>
</tr>
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<td>25</td>
<td>2.2</td>
<td>296</td>
<td>1.97</td>
<td>21.72</td>
<td>1.01</td>
</tr>
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<td>19</td>
<td>1.9</td>
<td>302</td>
<td>0.24</td>
<td>3.74</td>
<td>0.04</td>
</tr>
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<td>Colonial</td>
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<td>2.1</td>
<td>295</td>
<td>1.66</td>
<td>24.45</td>
<td>1.15</td>
</tr>
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<td>21</td>
<td>2.1</td>
<td>306</td>
<td>1.82</td>
<td>26.48</td>
<td>0.87</td>
</tr>
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</tr>
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<td>2.03</td>
<td>284.0</td>
<td>1.435</td>
<td>16.547</td>
<td>0.690</td>
</tr>
</tbody>
</table>

N = number of jaw specimens  
TNT = total number of teeth  
X = mean  
DMFS = decayed, missing, filled tooth surfaces  
DS = decayed surfaces
### TABLE 3
Frequency and Severity of Osteitis, Calculus and Bone Loss in the Permanent Dentitions

<table>
<thead>
<tr>
<th>Culture</th>
<th>OSTEITIS</th>
<th>CALCULUS % Affected</th>
<th>BONE LOSS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>% Affected</td>
<td>N</td>
</tr>
<tr>
<td>Paracas</td>
<td>maxillary</td>
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<td>32.6</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>31</td>
<td>38.7</td>
</tr>
<tr>
<td>Nazca</td>
<td>maxillary</td>
<td>9</td>
<td>44.4</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>20</td>
<td>45.0</td>
</tr>
<tr>
<td>Huari</td>
<td>maxillary</td>
<td>13</td>
<td>15.3</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>14</td>
<td>14.2</td>
</tr>
<tr>
<td>Ica</td>
<td>maxillary</td>
<td>24</td>
<td>16.7</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>25</td>
<td>20.0</td>
</tr>
<tr>
<td>Inca</td>
<td>maxillary</td>
<td>20</td>
<td>33.0</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>19</td>
<td>50.0</td>
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<td>maxillary</td>
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<tr>
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<td>mandibular</td>
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<td>23.8</td>
</tr>
<tr>
<td>Average</td>
<td>maxillary</td>
<td>22.0</td>
<td>27.00</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>21.7</td>
<td>31.95</td>
</tr>
</tbody>
</table>

N = number of jaw specimens
X = mean

### TABLE 4
Frequency of Missing or Unerupted Teeth in the Primary Dentitions

<table>
<thead>
<tr>
<th>Culture</th>
<th>TEETH LOST</th>
<th>X TEETH LOST PER JAW</th>
<th>% CONGENITALLY MISSING</th>
<th>MOLAR 1</th>
<th>MOLAR 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>NTP</td>
<td>NUMBER</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Paracas</td>
<td>maxillary</td>
<td>9</td>
<td>90</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>6</td>
<td>60</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Nazca</td>
<td>maxillary</td>
<td>2</td>
<td>20</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>2</td>
<td>20</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Huari</td>
<td>maxillary</td>
<td>4</td>
<td>40</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>mandibular</td>
<td>3</td>
<td>30</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Ica</td>
<td>maxillary</td>
<td>14</td>
<td>140</td>
<td>0</td>
<td>0.0</td>
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<td>mandibular</td>
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<td>150</td>
<td>2</td>
<td>1.3</td>
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<td>maxillary</td>
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<td>20</td>
<td>0</td>
<td>0.0</td>
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<tr>
<td></td>
<td>mandibular</td>
<td>3</td>
<td>30</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Colonial</td>
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<td>mandibular</td>
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<td>160</td>
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<td>0.0</td>
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<td>Average</td>
<td>maxillary</td>
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<td>76.7</td>
<td>0.16</td>
<td>0.12</td>
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<tr>
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<td>mandibular</td>
<td>7.5</td>
<td>75.0</td>
<td>0.33</td>
<td>0.22</td>
</tr>
</tbody>
</table>

N = number of jaw specimens
NTP = number of teeth possible
X = mean
TABLE 5
Frequency and Severity of Attrition and Dental Caries in the Primary Dentitions

<table>
<thead>
<tr>
<th>Culture</th>
<th>X Score of attrition 0,1,2,3</th>
<th>TNT</th>
<th>Caries Based on DMFS</th>
<th>Caries Based on DS</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>X DMFS/tooth</td>
<td>X DMFS/jaw</td>
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<td>Paracas</td>
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<td></td>
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<tr>
<td>maxillary</td>
<td>9</td>
<td>1.5</td>
<td>76</td>
<td>0.49</td>
</tr>
<tr>
<td>mandibular</td>
<td>6</td>
<td>1.3</td>
<td>54</td>
<td>0.35</td>
</tr>
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<td>Nazca</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>2</td>
<td>1.3</td>
<td>19</td>
<td>0.21</td>
</tr>
<tr>
<td>mandibular</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Huari</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>4</td>
<td>1.5</td>
<td>34</td>
<td>0.47</td>
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<td>3</td>
<td>1.6</td>
<td>24</td>
<td>0.17</td>
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<td></td>
</tr>
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<td>maxillary</td>
<td>14</td>
<td>1.7</td>
<td>105</td>
<td>0.47</td>
</tr>
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<td>mandibular</td>
<td>15</td>
<td>1.6</td>
<td>123</td>
<td>0.33</td>
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<tr>
<td>Colonial</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>2</td>
<td>1.0</td>
<td>10</td>
<td>0.10</td>
</tr>
<tr>
<td>mandibular</td>
<td>3</td>
<td>0.8</td>
<td>28</td>
<td>0.00</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>7.7</td>
<td>1.37</td>
<td>59.8</td>
<td>0.382</td>
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<td>1.28</td>
<td>71.4</td>
<td>0.242</td>
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</tbody>
</table>

N = number of jaw specimens
TNT = total number of teeth
X = mean
DMFS = decayed, missing, filled tooth surfaces
DS = decayed surfaces

Indians, the reasons for different caries incidence in the remaining groups are not as obvious. The three groups scoring high on caries also scored highest in calculus involvement. The Paracas, Ica and Colonial individuals who had the highest caries incidence were all members of “coastal” as opposed to “inland” cultures. Something in the diet or water of the “inland” people, such as fluoride, could have protected them against caries. Further investigation on fluoride content of bones and teeth should shed some light on this relationship. The reason for a lower incidence of caries in the Nazca individuals, also a “coastal” group, is not readily apparent.

The maxillae are more frequently involved with caries than the mandible. In the present study this was noted using both the incidence of caries based on DMFS and on DS in primary dentitions. The maxillae were shown to be more frequently involved with caries using the DS index in the permanent dentitions, but because of the large number of missing teeth (antemortem) in the mandibles of the permanent dentitions, the DMFS showed the incidence of caries to be about equal in the two arches. Because of the method of scoring using the DMFS index, this can be considered a misleading value and again the maxillae showed a higher incidence of caries. In this study, using optimal gross-examination coupled with roentgenographic analysis, 26.7% of the individuals with primary dentitions and 85.2% of the individuals with permanent dentitions had one or more carious lesions. In our earlier study 70% of the permanent mandibles had one or more carious lesions. Leigh reported a 35% incidence in specimens having both jaws. Although the reason is still not evident, the high incidence of caries has now been shown to be consistent in our two studies and in fact increased because of the inclusion in this present study of maxillae having a higher incidence of dental caries. The caries incidence in the combined adult population in the United States as of 1962 was 20.4 DMFS teeth per person. The overall average of DMFS per specimen in our earlier pre-Columbian study was 13.76 in 101
TABLE 6

Frequency and Severity of Osteitis, Calculus and Bone Loss in the Primary Dentitions

<table>
<thead>
<tr>
<th>Culture</th>
<th>Osteitis</th>
<th>Calculus % Affected</th>
<th>Bone Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>% Affected</td>
<td>N</td>
</tr>
<tr>
<td>Paracas</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>9</td>
<td>11.1</td>
<td>9</td>
</tr>
<tr>
<td>mandibular</td>
<td>6</td>
<td>16.7</td>
<td>6</td>
</tr>
<tr>
<td>Nazca</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>2</td>
<td>0.0</td>
<td>2</td>
</tr>
<tr>
<td>mandibular</td>
<td>2</td>
<td>0.0</td>
<td></td>
</tr>
<tr>
<td>Huari</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>4</td>
<td>0.0</td>
<td>4</td>
</tr>
<tr>
<td>mandibular</td>
<td>3</td>
<td>0.0</td>
<td>3</td>
</tr>
<tr>
<td>Ica</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>14</td>
<td>7.1</td>
<td>14</td>
</tr>
<tr>
<td>mandibular</td>
<td>15</td>
<td>6.7</td>
<td>15</td>
</tr>
<tr>
<td>Inca</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>2</td>
<td>0.0</td>
<td>2</td>
</tr>
<tr>
<td>mandibular</td>
<td>3</td>
<td>0.0</td>
<td>3</td>
</tr>
<tr>
<td>Colonial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>15</td>
<td>6.7</td>
<td>15</td>
</tr>
<tr>
<td>mandibular</td>
<td>16</td>
<td>12.5</td>
<td>16</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maxillary</td>
<td>7.7</td>
<td>4.15</td>
<td>7.7</td>
</tr>
<tr>
<td>mandibular</td>
<td>7.5</td>
<td>5.98</td>
<td>8.6</td>
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</table>

N = number of jaw specimens  
X = mean

permanent mandibles. In the present study the average DMFS was 16.3 per maxillary specimen and 16.5 per mandibular specimen. The higher DMFS per specimen in the present study as compared to our earlier work is no doubt related to the use of the x-ray unit. While these figures do not correspond exactly with those in the United States as of 1962, they are similar, as to incidence of caries, to the present study. Caries data on present day Peruvians are not available for comparison.

Osteitis was noted in all six cultures. The Nazca and Inca showed the highest incidence in the arches with permanent dentitions while the Paracas, Ica and Colonial individuals had the highest incidence in the primary. This finding paralleled the findings on the mean number of missing antemortem teeth while calculus accumulation appeared to relate directly to caries incidence.

The amount of alveolar bone loss was moderate among individuals from all cultures with those from the Paracas culture leading the way. Bone loss did not correlate directly with calculus or caries scores. It is plausible that a less refined and abrasive diet caused both moderate attrition and alveolar bone loss in all cultures irrespective of age.

REFERENCES


Volume Fourteen

TABLE OF CONTENTS

1978 • Number One

Paleoepidemiology

Introduction 2

Patterns of Prehistoric Epidemiology and Human Paleopathology 3
MAHMOUD Y. EL-NAJJAR, PH.D.

Porotic Hyperostosis in the Eastern Mediterranean 10
J. LAWRENCE ANGEL, PH.D.

Paleoepidemiology of Infectious Disease in the Dickson Mounds Population 17
JOHN LALLO, PH.D.
GEORGE J. ARMELAGOS, PH.D.
JEROME C. ROSE, PH.D.

Yawslike Disease Processes in a Louisiana Shell Mound Population 24
LOUISE M. ROBBINS, PH.D.

Pre-Columbian Tuberculosis: An Epidemiological Approach 32
JANE E. BUIKSTRA, PH.D.
DELLA C. COOK, PH.D.

Paleoepidemiology of Degenerative Joint Disease 45
ROBERT D. JURMAIN, PH.D.

Book Review 57

Appropriate Antibiotic Therapy for Urinary Tract Infections 69
SHELDON M. MARKOWITZ, M.D.

Intrascrotal Masses: Differentiation, Diagnosis, and Management 76
STEPHEN N. ROUS, M.D.

Common Pediatric Problems: Hypospadias, Enuresis, and Circumcision 77
JOHN H. TEXTER, JR., M.D.

Management of Carcinoma of the Kidney and Urinary Bladder 80
WARREN W. KOONTZ, JR., M.D.

Testicular Carcinomas and Carcinoma of the Prostate 83
PAUL F. SCHHELLHAMMER, M.D.

Adenocarcinoma of the Prostate: The Rationale and Role for Radiotherapy in its Management 88
TAPAN A. HAZRA, M.D.

Management of Acute Glomerulonephritis 90
DONALD OKEN, M.D.

The Management of End-Stage Renal Disease (ESRD) 92
WILLIAM F. FALLS, JR., M.D.

Male Infertility: The Clinical Aspects of Evaluation and Management 96
J. WILLIAM McROBERTS, M.D.

Scripta Medica

Maxillary and Mandibular Jaw Size in Pre-Columbian Peru 101
DANNY R. SAWYER, D.D.S., PH.D.
MARVIN J. ALLISON, PH.D.
RICHARD P. ELZAY, D.D.S., M.S.D.
DENNIS G. PAGE, D.D.S., M.S.
ALEJANDRO PEZZIA, PH.D.
1978 • Number Three

Kidney Disease

Introduction 110

Cancer and the Kidney
DONALD E. OKEN, M.D. 111

Glomerulonephritis
WILLIAM F. FALLS, JR., M.D. 116

Management of the Nephrotic Syndrome
DOUGLAS M. LANDWEHR, M.D. 124

On the Prevention of Acute Renal Failure
(Vasomotor Nephropathy)
DONALD E. OKEN, M.D. 128

Pyelonephritis
WILLIAM F. FALLS, JR., M.D. 132

Evaluation of an Abnormal Urinalysis in the
Asymptomatic Patient
DOUGLAS M. LANDWEHR, M.D. 140

The End-Stage Renal Disease Program (Adapted
from Treatment of End-Stage Renal Disease)
WILLIAM K. STACY, M.D. 144

Diabetic Nephropathy
BARRY B. KIRSCHBAUM, M.D. 148

1978 • Number Four

Introduction 158

Respiratory Therapy Modalities in the Treatment of
Acute Respiratory Failure
JAMES A.L. MATHERS, JR., M.D. 159

Screening Pulmonary Function Tests
GEORGE W. BURKE, III, M.D. 163

Flexible Fiberoptic Bronchoscopy
ORHAN MUREN, M.D. 169

Bedside Flow-Directed Balloon Catheterization in
the Critically Ill Patient
J. EUGENE MILLEN
FREDERICK L. GLAUSER, M.D. 172

Intrapulmonary Lymph Node Presenting as a ‘Coin’
Lesion: A Case Report
SIHU P. SAHA, M.D.
PORTER MAYO, M.D. 176

Maxillary Periapical Actinomycosis: A Case with an
Unusual Roentgenographic Appearance
DANNY R. SAWYER, D.D.S., Ph.D.
DENNIS G. PAGE, D.D.S., M.S. 178

The Dental Health Status of Pre-Columbian
Peruvians: A Study of Dental Caries, Missing
Teeth, Attrition, Osteitis, Calculus, and Bone Loss
DANNY R. SAWYER, D.D.S., Ph.D.
MARVIN J. ALLISON, Ph.D.
RICHARD P. ELZAY, D.D.S., M.S.D.
ALEJANDRO PEZZIA, Ph.D. 181

TABLE OF CONTENTS FOR VOLUME FOURTEEN 189

INDEX TO VOLUME FOURTEEN 191
Volume Fourteen
AUTHOR INDEX

ALLISON, MARVIN J., Ph.D., 101, 181
ANGEL, J. LAWRENCE, Ph.D., 10
ARMELAGOS, GEORGE J., Ph.D., 17

BUKSTRA, JANE E., Ph.D., 32
BURKE, GEORGE W., III, M.D., 163

COOK, DELLA C., Ph.D., 32

ELZAY, RICHARD P., D.D.S., M.S.D., 101, 181
EL-NAJJAR, MAHMOUD Y., Ph.D., 3

FALLS, WILLIAM F., Jr., M.D., 92, 116, 132

GLAUSER, FREDERICK L., M.D., 172
HAZRA, TAPAN A., M.D., 88
JURMAIN, ROBERT D., Ph.D., 45
KIRCHBAUM, BARRY B., M.D., 148
KOONTZ, WARREN W., Jr., M.D., 64, 80

LALLO, JOHN, Ph.D., 17
LANDWEHR, DOUGLAS M., M.D., 124, 140

MARKOWITZ, SHELDON M., M.D., 69
MASTERS, JAMES A. L., Jr., M.D., 159
MAYO, PORTER, M.D., 176
MCROBERTS, J. WILLIAM, M.D., 96
MILLEN, J. EUGENE, 172
MUREN, ORHAN, M.D., 169

OKEN, DONALD E., M.D., 90, 111, 128
PAGE, DENNIS G., D.D.S., M.S., 101, 178
PEZZIA, ALEJANDRO, Ph.D., 101, 181

ROBBINS, LOUISE M., Ph.D., 24
ROSE, JEROME C., Ph.D., 17
ROUS, STEPHEN N., M.D., 65, 76

SABA, SIBU P., M.D., 176
SAYYER, DANNY R., D.D.S., Ph.D., 101, 178, 181
SCHELLHAMMER, PAUL F., M.D., 83
STACY, WILLIAM K., M.D., 144
TEXTER, JOHN H., M.D., 77
# Volume Fourteen

## SUBJECT INDEX

<table>
<thead>
<tr>
<th>ACTINOMYCOSIS</th>
<th>pyelonephritis, 132</th>
</tr>
</thead>
<tbody>
<tr>
<td>maxillary and mandibular jaw size in pre-columbian peru, 101</td>
<td>the end-stage renal disease program, 144</td>
</tr>
<tr>
<td>bladder</td>
<td>management of end-stage renal disease (esrd), 92</td>
</tr>
<tr>
<td>cancer</td>
<td>lung disease</td>
</tr>
<tr>
<td>adenocarcinoma of the prostate: the rationale and role for radiotherapy in its management, 88</td>
<td>beside flow-directed balloon catheterization in the critically ill patient, 172</td>
</tr>
<tr>
<td>bronchoscopy</td>
<td>flexible fiberoptic bronchoscopy, 169</td>
</tr>
<tr>
<td>laryngectomy with an unusual roentgenographic appearance, 178</td>
<td>intrapulmonary lymph node presenting as a 'coin' lesion: a case report, 176</td>
</tr>
<tr>
<td>maxillary periapical actinomycosis: a case with an unusual roentgenographic appearance, 178</td>
<td>flexible fiberoptic bronchoscopy, 169</td>
</tr>
<tr>
<td>the dental health status of pre-columbian peruvians: a study of dental caries, missing teeth, attrition, osteitis, calculus, and bone loss, 181</td>
<td>intrapulmonary lymph node presenting as a 'coin' lesion: a case report, 176</td>
</tr>
<tr>
<td>cancer and the kidney, 111</td>
<td>intrapulmonary lymph node presenting as a 'coin' lesion: a case report, 176</td>
</tr>
<tr>
<td>management of carcinomas and carcinomatoses of the prostate, 83</td>
<td>intrapulmonary lymph node presenting as a 'coin' lesion: a case report, 176</td>
</tr>
<tr>
<td>CIRCUMCISION (see PEDIATRICS)</td>
<td>screening pulmonary function tests, 163</td>
</tr>
<tr>
<td>DENTISTRY</td>
<td>scrotum</td>
</tr>
<tr>
<td>maxillary and mandibular jaw size in pre-columbian peru, 101</td>
<td>intrascrotal masses: differentiation, diagnosis and management, 76</td>
</tr>
<tr>
<td>GLOMERULONEPHRITIS</td>
<td>testes</td>
</tr>
<tr>
<td>adenocarcinoma of the prostate: the rationale and role for radiotherapy in its management, 88</td>
<td>intrascrotal masses: differentiation, diagnosis, and management, 76</td>
</tr>
<tr>
<td>. . . , 116</td>
<td>testicular carcinomas and carcinoma of the prostate, 83</td>
</tr>
<tr>
<td>management of acute . . . , 90</td>
<td>tuberculosis</td>
</tr>
<tr>
<td>HYPOSPADIAS (see PEDIATRICS)</td>
<td>pre-columbian . . . : an epidemiological approach, 32</td>
</tr>
<tr>
<td>INFERTILITY</td>
<td>urethritis</td>
</tr>
<tr>
<td>male . . . : the clinical aspects of evaluation and management, 96</td>
<td>the female urethral syndrome and . . . and prostatitis in the male, 65</td>
</tr>
<tr>
<td>KIDNEY DISEASE</td>
<td>UROLOGY</td>
</tr>
<tr>
<td>cancer and the kidney, 111</td>
<td>adenocarcinoma of the prostate: the rationale and role for radiotherapy in its management, 88</td>
</tr>
<tr>
<td>diabetic nephropathy, 148</td>
<td>appropriate antibiotic therapy for urinary tract infections, 69</td>
</tr>
<tr>
<td>glomerulonephritis, 116</td>
<td>common pediatric problems: hypospadias, enuresis, and circumcision, 77</td>
</tr>
<tr>
<td>management of acute glomerulonephritis, 90</td>
<td>intrascrotal masses: differentiation, diagnosis, and management, 76</td>
</tr>
<tr>
<td>management of carcinoma of the kidney and urinary bladder, 80</td>
<td>management of carcinoma of the kidney and urinary bladder, 80</td>
</tr>
<tr>
<td>management of the nephrotic syndrome, 124</td>
<td>testicular carcinomas and carcinoma of the . . . , 83</td>
</tr>
<tr>
<td>on the prevention of acute renal failure (vasomotor nephropathy), 128</td>
<td>the female urethral syndrome and urethritis and prostatitis in the male, 65</td>
</tr>
<tr>
<td>PEDIATRICS</td>
<td>URINALYSIS</td>
</tr>
<tr>
<td>common pediatric problems: hypospadias, enuresis, and circumcision, 77</td>
<td>evaluation of an abnormal . . . in the asymptomatic patient, 140</td>
</tr>
<tr>
<td>PROSTATE</td>
<td>YAWS (see PALEOEPIDEMIOLOGY)</td>
</tr>
<tr>
<td>adenocarcinoma of the . . . : the rationale and role for radiotherapy in its management, 88</td>
<td></td>
</tr>
</tbody>
</table>
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MAY 1979

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