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OSTEOPATHIC MEDICINE

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Pneumonia is defined as "inflammatory disease of lung parenchyma." The terms pneumonia and pneumonitis are synonymous, though at the instance of clinician, pathologist, and roentgenologist alike, pneumonitis has of late acquired a limitation in meaning. The as yet only partly understood pathologic physiology of pneumonia together with the universal confusion of terminology and classification of the disease is unquestionably of major concern to the roentgenologist.

It was in 1819 that Laennec announced the basic or pathologic concept of pneumonia. The tissue changes have been denoted by a standard classification as the stages of pneumonic engorgement, hepatization, and resolution. All of these stages in this pathologic classification of pneumonia may prevail simultaneously in one lobe or different portions of the lung.

For many years an anatomic as well as a clinical classification of pneumonia has been that of lobar, croupous, or fibrinous pneumonia, and lobular, catarrhal, or bronchopneumonia. During recent years the increased knowledge of bacteriologic causes of pneumonia has subjugated both the aforementioned pathologic and anatomic classifications. Hence, at present writing, the etiologic classification is in vogue with identification of approximately fifty forms of pneumonia. This etiologic classification, which for the major part though not in entirety is bacteriological, is of definite importance to the roentgenologist who, serving as a consultant, must base his diagnostic opinion upon both clinical and film data.

Of the thirty-two classified types of pneumococci the roentgenologist does not profess to designate the type of offending organism. It has been established and reported by Reimann that pneumococcus types I, II, and XIV almost always cause lobar pneumonia, while types III, V, VII, and VIII and most of the higher types cause the lobar form in from 50 to 80 per cent of cases. A study of pathogenesis has indicated that pneumonia due to pneumococci of types I and II is an exogenous infection, while pneumonia attributable to the pneumococci of the types III to XXXII is usually an autogenous infection. Diagnostically and therapeutically, it is of importance to note that type II is the most virulent of all pneumococci.

In 1925 Belden reviewed a series of 357 cases of typed pneumococcal lobar pneumonia. Special attention was directed to the anatomic or lobe
distribution of the different types of pneumococci. It is an established fact that in the adult the tubercle bacillus has a special predilection for the upper lobe of the lung, and hence Belden's investigation was undertaken to determine whether different types of pneumococci has a predilection for specific lobes of the lung. His observations or conclusions, although interesting, do not justify diagnosis of type of invading pneumococcus by x-ray examination.

Bronchopneumonia, which is decidedly different from the lobar form both clinically and radiologically, has in most instances been found to be caused by the higher numbered types of pneumococci, hemolytic streptococci, staphylococci, or influenza bacilli.

A concise clinical and roentgenologic classification of pneumonia was quite recently suggested by Cole, and has been tentatively accepted by most authorities. He advocates that all pneumonias be classified as "typical" or "atypical." The "typical" pneumonia is lobar pneumonia, and all pneumonias not conforming to the lobar type have been relegated to the class of "atypical" pneumonia.

"Typical" or Lobar Pneumonia

In a monograph prepared by the Rockefeller Institute (Avery, Chickering, Cole, Dochez), it is recorded that lobar pneumonia is almost always caused by the pneumococcus. This pulmonary infection possesses a fairly typical clinical course, and the roentgen manifestations of the disease are quite constant.

Film evidence of lobar pneumonia will usually be present six to eighteen hours following the onset of symptoms, and Rigler states that absence of radiographic findings twenty-four hours after the onset of symptoms is strong evidence against the presence of lobar pneumonia.

The initial pathologic state of congestion will be manifest by a prominence of the peribronchial vascular relief which may prevail for only a few hours. Other early film findings are described as a diffuse hazing throughout the involved lobe, a somewhat irregular ovoid opacity situated in the hilar zone of the lung, or a triangular zone of increased parenchymal density, the base of which is located at the lung periphery with the apex directed and extending toward the hilum.

The major pathologic involvement is that of an exudation with inflammatory edema rich in pneumococci within the alveoli. It is believed that this process spreads to other portions of the lobe by direct extension from alveolus to alveolus and also to other lobes of the lung by the transportation of infected edema fluids by way of the bronchi. The parenchymal consolidation as demonstrated by the roentgenogram is attributable to the alveolar exudation displacing the air content and filling the lumen with a fibrinous liquid which soon becomes a clotted mass of solid consistency.
X-RAY DIAGNOSIS IN PNEUMONIA

Sante is of the opinion that adult lobar consolidation usually begins in the region of the hilum and extends toward the periphery, while Rigler and others have recorded the initial lung consolidation to be more common at the periphery of the lobe.

The pathologic physiology of this disease is subject to many variations, and it is emphasized by Rigler that consolidation of a whole lobe is not a necessary feature of lobar pneumonia. The consolidation of lobar pneumonia progresses rapidly, and a complete lobar opacity is often present at the time of the first film study.

It is generally advocated that serial film study with frequent intervals offers an unexcelled method for premortem study of the gross pathology of pneumonia. Radiographic investigation utilizing two planes or projections is obviously the procedure of choice, though the physical status of the patient many times only permits of a single anterior or posterior chest film secured by means of the portable unit.

Complete lobar consolidation represented by a homogeneous opacity devoid of bronchovascular reticulation is notably well demarcated at the respective interlobar fissure. The anatomic distribution of the lobes or topography of the lung determines the roentgenologic characteristics of lobar consolidation. Involvement or consolidation of the upper lobe of the right lung will be sharply demarcated at the inferior margin of the lobe which horizontally traverses the midchest while the extreme apex of the lobe is often not involved. A middle lobe pneumonic opacity which is relatively small will exhibit a horizontal, discrete, and straight upper margin traversing the chest at the interlobar fissure, while the lower border often appears irregular and less dense, blending with dependent normal lung tissue. In the lateral film, this middle lobe consolidation is located in the anterior one-half of the chest and is triangular in shape with the base of the triangle abutting the anterior chest wall.

In the anterior or posterior film, a lower lobe consolidation on the right side will obscure the middle lobe area with the upper margin of this parenchymal opacity ill defined and its lower border blending with the density of the diaphragmatic leaflet. In contradistinction to the findings prevailing in the presence of a free pleural exudate, it is well to emphasize that in lower lobe pneumonia the costophrenic angle is the last portion of the lobe to exhibit pneumonic consolidation. Due to the oblique plane of the interlobar fissure, a consolidation of either the upper or lower lobe of the left lung will present no abrupt demarcation at the respective lobar division except as visualized in lateral projection.

Complete consolidation of a whole lung is not infrequent. Such a pneumonic state in most instances is distinguished from a pleural effusion only by the absence of a mediastinal shift to the contralateral side.

The experimental work of Blake and Cecil on the pathogenesis of pneumonia indicates that the pneumococci, when reaching the larger bronchial divisions by way of the upper respiratory tract and trachea, pene-
trate the walls of the bronchi and progress toward the alveoli and lung periphery in the peribronchial interstitial connective tissue.

In 1929 a notable publication by Coryllos and Birnbaum stated that atelectasis and pneumonia have a common pathogenesis. They concluded that the bronchi were first involved with an inflammatory exudate resulting in bronchial obstruction and subsequent lobular or lobar atelectasis followed by parenchymal inflammatory pathology. This experimental work was further qualified with regard to lobar and lobular atelectasis by stating that the offending organism producing much edema or thick exudate would occlude the larger bronchi. Most of the authoritative writings refer to the work of Coryllos and Birnbaum though these writings conclude that the meaning of the term atelectasis has been extended beyond the accepted criteria, and that a relationship may exist between atelectasis and pneumonia though they are basically different pathologic states. Roentgenologically, a restriction of diaphragmatic mobility prevails on the side of pneumonic involvement; however, narrowing of intercostal spaces and a mediastinal shift, the salient features of atelectasis, are not observed in lobar pneumonia.

In accordance with the physical findings, the lobar consolidation shows no radiographic change at the time of crisis, though within twenty-four hours there is usually some evidence of resolution. The process of resolution is uniformly quite rapid, occurring within three to fourteen days. The density of the consolidated lobe gradually diminishes, and there are observed concomitant multiple irregular areas of parenchymal ventilation. Finally, only a residual exaggeration of the relief of the regional truncal or bronchovascular network is noted, and this may persist for a considerable time after complete resolution.

The bulk of the exudate is believed to be dissolved and resolved. During serial film surveillance of cases of lobar pneumonia receiving radiation therapy, Lloyd has observed a post-therapeutic diffuse clouding or veiling of adjacent uninvolved portions of the lung which, upon first evaluation, may suggest lobar progression of the pathology. To the best of my knowledge no mention of this observation has been recorded in the literature. Lloyd has concluded that this phenomena may in part at least be attributed to the resorption of the inflammatory alveolar exudate by way of the pleural lymphatics. This latter observation apparently has no direct relationship to the "protective fluid" separating the inflamed pleural surfaces in many cases of lobar pneumonia.

Pleural thickening is a relatively common sequela to lobar pneumonia, while pleural effusion or empyema constitute the more common complications. Resolution may be delayed, though in the absence of progressive favorable film findings at the end of a three week interval one is justified in suspecting a complicating pathology. Non-resolution followed by chronic fibrous or interstitial pneumonia may be characterized by a replacement of the exudate with fibrous connective tissue.
In 1837 Seifert introduced the term bronchopneumonia to describe an inflammation of the lung manifest by multiple focal or diffuse lesions originating in the bronchioles and extending to involve the pulmonary lobules and parenchyma. The multitude of synonyms in the literature of today, together with the great variability of etiology, clinical signs, symptoms, and roentgenologic manifestations of bronchopneumonia warrants the more applicable term "atypical" pneumonia. For purposes of clarity, atypical pneumonia has been divided into two main groups according to mode of origin. The first group or primary form includes all atypical pneumonias common to patients previously well or without a predisposing illness, such form being exogenous in type and common to the extremes of life when the powers of resistance are either poor or not well developed, as in the infant. The secondary form of atypical pneumonia is described by Reimann as a pulmonary infection which occurs during, or after some other infectious or debilitating disease, or some mechanical or chemical injury to the lungs with the infection thereby usually endogenous in character.

The early roentgenologic manifestations of the "garden variety" of bronchopneumonia, or better, primary atypical pneumonia, are an increased size and radiodensity of the pulmonary hila with prominence of the bronchovascular reticular pattern and slight mottling of both lungs. Small, soft infiltrations or patchy areas of increased radiodensity, variable in size and poorly demarcated or hazed as to marginal relief, are subsequently observed distributed about or along the course of the bronchovascular network. Sante emphasizes that these irregular areas of consolidation are usually distributed about the bronchi of the lower lobes. This apparent lobular arrangement of parenchymal opacities may, by virtue of coalescence, assume a regional pseudo-lobar distribution. Of differential diagnostic importance is the fact that the tissue changes, though variable as to degree, will be multilobar and bilateral. A concomitant hilar and peritracheal adenopathy is common, particularly in children.

Resolution of the pathology is characteristically slow, often requiring from four to six weeks; while the most common complication is pulmonary abscess. It is to be recorded that the primary atypical streptococcus and staphylococcus pneumonias possess a rather high frequency of complicating empyema.

Cole and Reimann together with many internists and radiologists have in the past few years encountered an, in part, epidemic form of primary atypical pneumonia the age incidence of which is unqualified and the exact etiology of which is unknown. The highly contagious nature of this pneumonia has led pathologists to suspect a virus etiology. At the present time the status of laboratory investigative work warrants replacement of the term virus pneumonia by the classification "primary atypical pneumonia, etiology unknown." Although it has been impossible to obtain
accurate statistics, it is believed this disease has outnumbered proven pneumococcus pneumonias by four to one. Special attention of the internists was initiated by the non-seasonal occurrence and relatively benign nature of the disease, and as well by the absence in the sputum of characteristic bacterial agents in sufficient numbers to be etiologically significant. Rhoads likewise states that observations of major clinical importance are the absence of prostration as compared to the amount of pulmonary involvement manifest by roentgenograms, a normal or low leukocyte count in uncomplicated cases, and the relatively low respiratory rate.

The radiologist has had no small part in the recognition and study of atypical pneumonia of undetermined etiology. Film observations have been of prime importance in demonstrating the great variability in the type of lesion and the interstitial features of the disease.

Saphir described the pathology of primary atypical pneumonia of undetermined etiology as one of inflammation extending from the peribronchial and perivascular lymphatics into the interalveolar tissues with secondary involvement of respective alveoli. Both radiographically and pathologically, the term “pneumonitis” has been reserved to designate an interstitial pneumonia; hence, in a film description of the pathology in question, we may employ the term “acute pneumonitis.”

The radiographic characteristics common to this primary atypical pneumonia of undetermined etiology have been well presented by both Kornblum and McCarthy. Cases of one group have exhibited a localized area of increased parenchymal density which occurs predominantly in the lower lobes. This opaque zone varied considerably in size and radiodensity. However, the density was never found to be as great as that noted in the consolidation of lobar pneumonia. The aforementioned pathology may designate the entire extent of the disease, while in some instances this opacity constitutes a focus with the inflammatory pathology spreading to involve a large portion of the lung.

In another group, the lesion appears as a patchy or lobular involvement confined to a lobe, with confluence of the consolidative opacities. In the main, the appearance is suggestive of the bronchopneumonic form of atypical pneumonia. A subsequent multilobar or bilateral involvement may appear.

Further observations indicate a group in which the pathology is manifest in terms of a bronchovascular reticular distribution of multiple confluent parenchymal radiopacities radiating from a prominent hilum to involve in part or in entirety one lobe of the lung. When either upper lobe is the site of this type of lesion, all authorities are in agreement that only serial film investigation will distinguish primary atypical pneumonia of unknown origin from tuberculosis.

Kornblum has recorded several cases with pathology exhibiting a predilection for the hilar region, unilateral or bilateral, the pneumonia simulating that encountered in epidemic influenza.
A clearing of the pulmonary opacities is usually observed within five to ten days; however, the disease may be protracted over a period of two to three weeks. As in other forms of pneumonia, the roentgen changes often persist after apparent recovery of the patient. No pulmonary or pleural complications have been encountered, though Rhoads is of the opinion that bacterial complications may become more frequent in the cases of primary atypical pneumonia of unknown etiology.

In a paper pertaining to atypical pneumonia, it is mandatory that one make mention of Friedlander bacillus pneumonia and lipoid pneumonia.

The onset of Friedlander bacillus pneumonia is often acute and clinically similar to that of lobar or "typical" pneumonia, while of differential diagnostic importance are the clinical course and roentgenologic characteristics of the disease. Film study of the chest will not infrequently display unilateral distribution of large pneumatic opacities which coalesce with a resultant pseudo-lobar consolidation. Roentgenographic demonstration of rapid tissue destruction and abscess formation within the area or areas of parenchymal consolidation and an exceedingly high mortality rate are notable characteristics of Friedlander bacillus pneumonia.

Lipoid pneumonia, a non-bacterial atypical pneumonia attributable to the aspiration of various kinds of oil, has been observed in all age groups, though this specific form of pneumonia is more common in the infant. Both clinically and roentgenologically the pneumatic pathology involves the basal or lower portion of the lung; and while often bilateral, the parenchymal consolidation will be predominant on the right side. The radiographic manifestations are lipoid pneumonia, which are usually in excess of the physical signs of the disease, are variable and in part dependent upon the degree of secondary infection prevailing at time of study.

In concluding this short discussion of the as yet only partly understood pathologic state, pneumonia, it should be emphasized that the roentgenologist serving as a consultant must interpret the film evidence of this or any disease only when cognizant of the clinical status of the patient.

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A REVIEW OF RESEARCH

II
THE EFFECT OF CERTAIN TYPES OF MANIPULATION ON
BLOOD PRESSURE AND PULSE RATE IN
COLLEGE STUDENTS

FREDERICK A. LONG

Director of Research

In the introduction to this review certain major research series were listed. One of these had to do with the effect of various physical factors on certain measurable physiological processes. Several studies in this series have been reported during the period covered by this review, having to do with the effects of specific types of manipulation on blood pressure and pulse rate. A composite analysis of the results of these studies will be reported here.

Experiments were carried out for the general purpose of observing the effects on blood pressure and pulse rate produced by various specific types of osteopathic manipulation applied to non-clinical subjects. One of the requirements of scientific procedure is that it isolate elements, treat each element separately, and then correlate the results. Some of our previous attempts to generalize about the effects of manipulation have violated a principle of science by combining distinctive elements into a whole picture and talking about the picture without first understanding the elements. We should attempt to have rather exact information concerning the effects of individual types of manipulation to be in position to explain the effects of "osteopathic manipulative treatment."

The experiments to be reported utilized the following techniques: Sudden spinal joint mobilization in the cervical, upper thoracic, lower thoracic and lumbar, and entire spinal areas; pressure in the suboccipital and upper thoracic spinal areas; soft tissue manipulation in the cervical spinal area; and a combination of cervical soft tissue manipulation and sudden cervical spinal joint mobilization.

The source material for these experiments was a group of non-clinical male students of the Philadelphia College of Osteopathy. Their age distribution, and original blood pressure and pulse rate readings are shown in table 1. One-half the number of students constituted subjects receiving manipulation, and the other half controls. A comparison of these groups is also shown in table 1.

Certain conditions of the experiments were identical throughout. All experiments were carried out in the Department of Osteopathic Research at the Philadelphia College of Osteopathy. The work was done between the hours of 1:00 and 4:00 p.m. in a well-ventilated room kept at even
Table I
Average and extreme initial ages, blood pressures, and pulse rates in subjects and controls.

<table>
<thead>
<tr>
<th></th>
<th>Subjects</th>
<th></th>
<th>Controls</th>
</tr>
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<tbody>
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<td>av.</td>
<td>range</td>
<td>av.</td>
</tr>
<tr>
<td>Age</td>
<td>22.8</td>
<td>17-46</td>
<td>22.7</td>
</tr>
<tr>
<td>Systolic</td>
<td>113.3</td>
<td>74-168</td>
<td>115.6</td>
</tr>
<tr>
<td>Diastolic</td>
<td>71.1</td>
<td>40-102</td>
<td>73.0</td>
</tr>
<tr>
<td>Pulse</td>
<td>73.5</td>
<td>52-107</td>
<td>72.3</td>
</tr>
</tbody>
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temperature. All subjects and controls were made fully aware of the nature of the experiment to be carried out. No talking was permitted on the part of subjects and controls after the experiments began, and only occasionally was it necessary for the operators to converse. Sphygmomanometers used in the experiments on cervical mobilization and upper thoracic mobilization were of the spring type (Tycos). In all other experiments, mercury type sphygmomanometers were used. The cuff was applied to the left arm and the auscultatory method used in determining blood pressure. In determining pulse rate, the radial pulse at the left wrist was counted for one-half minute.

One subject and one control were studied at each experiment; therefore, there were four persons present in the laboratory, one subject, one control, and two operators.

An approximately uniform amount of clothing was worn by all subjects and controls, and constricting bands were released. Ordinary straight-type osteopathic treatment tables were used throughout.

Cervical Spinal Mobilization

One hundred male students were used to observe the effects of sudden cervical spinal mobilization on blood pressure and pulse rate. Fifty were used as subjects receiving manipulation, and fifty as controls. The average age of the subjects was 25.8 years, and of the controls 21.3 years.

Subjects and controls assumed the supine position and rested for five minutes, at the end of which time systolic and diastolic blood pressures and pulse rates were recorded. In the subjects, sudden mobilization of the cervical spinal joints was produced by the following technique: A fixed point was established by the operator's index finger on one side of the neck posterolaterally at the level of the third cervical vertebra. Rotation
of the head and cervical column was produced away from the side of fixation, with slight extension and side bending toward the fixed side. Rotation was carried to its limit; and when this point was reached, exaggeration of all movements was carried out by a slight thrust with the index finger at the third cervical vertebra and counter-force by the operator's hand placed on the opposite side of the subject's head. In most instances, there occurred the series of characteristic "pops" to be heard when joints are suddenly mobilized. The entire procedure was repeated on the opposite side, both sides being mobilized within a period of about five seconds.

Systolic and diastolic blood pressures were determined in subjects and controls immediately after mobilization in the subjects. Pulse rates were determined immediately after blood pressures were taken. Subjects and controls rested in the supine position for three minutes, and the blood pressures and pulse rates were again taken.

**Upper Thoracic Spinal Mobilization**

Upon completion of the previous cervical mobilization, these subjects and controls assumed the sitting position and rested for one minute, at the end of which time blood pressures and pulse rates were again recorded.

Mobilization of the upper thoracic spinal joints was carried out as follows: The subject clasped his hands behind his neck. The operator, standing behind the subject, passed his hands in front of the subject's arms and clasped them over the subject's hands behind the subject's neck. A small pillow was placed between the operator's chest and the subject's mid-thoracic vertebral area. The subject was instructed to let his head and body drop into a relaxed position and to fall backward toward the operator who maintained flexion tension on the subject's upper thoracic spine. The subject's cervical column was completely immobilized by the subject's and operator's hands. At the limit of flexion tension in the spine, the operator suddenly exaggerated the position of flexion to produce sudden mobilization of the upper thoracic spinal articulations. The controls were carried through the same movements as the subjects except that the final sudden mobilization was not produced. Immediately after the above procedure, the blood pressures and pulse rates were recorded for both subject and control. Subject and control then remained in the sitting position for three minutes, at the end of which time blood pressures and pulse rates were again determined and recorded.

**Lower Thoracic and Lumbar Spinal Mobilization**

Seventy-six male students were used to observe the effects of sudden spinal mobilization in the lower thoracic and lumbar areas on blood pressure and pulse rate. Thirty-eight were used as subjects receiving manipulation and thirty-eight as controls. The average age of the subjects was 23.1 years and of the controls 23.0 years.
Subjects and controls assumed the supine position and rested for ten minutes, at the end of which time blood pressures and pulse rates were recorded. In the manipulated subject, the following procedure was carried out immediately after the original readings were made: The subject turned to the left lateral position, the under-arm was drawn forward, and the right knee was drawn upward and flexed to a position resting on the table in front of and above the left. Forward pressure was applied to the right hip and backward counter-pressure applied to the right shoulder. When the limit of motion was reached, sudden mobilizing force was brought to bear upon the lumbar and lower thoracic vertebrae. The characteristic “popping” sound frequently heard when spinal joints are suddenly mobilized was elicited in the majority, but not in all of the subjects manipulated. After mobilization had been carried out on one side, the subject was instructed to turn slowly to the opposite side and the process was repeated. The control made all movements carried out by the subject, but no mobilizing forces were applied. Immediately after completion of mobilization, the subject and control returned to the supine position; and the blood pressures and pulse rates were immediately determined. This concluded the experiment.

Mobilization of the Entire Spine

Sixty male students were used to study the effects on blood pressure and pulse rate of sudden mobilization of the entire spine. Thirty were used as subjects for manipulation and thirty as controls. The average age of the subjects was 24.9 years and of the controls 24.5 years.

Subject and control assumed the supine position and rested for ten minutes, after which time blood pressures and pulse rates were recorded. In the manipulated subject, the following procedure was carried out: The subject turned to the left lateral position, the under-arm was drawn forward, and the right knee was drawn up and flexed to a position resting on the table in front of and above the left knee. Forward pressure was applied to the right hip and backward counter-pressure applied to the right shoulder. When the limit of motion was reached, sudden mobilizing force was brought to bear upon the lumbar and lower thoracic spine. The characteristic “popping” sound heard when joints are suddenly mobilized was elicited in the majority, but not in all of the subjects manipulated. After mobilization had been carried out on one side, the subject turned slowly to the opposite side and the procedure was repeated. The subject then turned to the supine position, with hands clasped behind his neck, and with elbows comfortably approximating in front. The operator, standing at the right of the subject, grasped the subject’s elbows and produced flexion and right rotation of the thoracic spine. The operator’s folded right hand was then placed under the mid-thoracic spine on both sides of the spinous processes and the subject’s body brought vertically
over the operator's hand with flexion still maintained. Mobilization of the mid-thoracic spine was produced by maintaining downward pressure on the subject's elbows and at the same time allowing the spine to fall toward extension. This procedure was repeated with the operator's right hand at a higher level to mobilize the upper thoracic segments. With the subject still resting supine, the operator took a position at the subject's head. Cervical mobilization was then produced in the following manner: A fixed point was established by the operator's index finger at one side of the neck at the level of the third cervical vertebra. The head and cervical column were rotated away from the side of fixation, and side-bending toward the side of the fixed point was produced. The cervical column was also carried into extension. Rotation was carried to its limit; and when this point was reached, exaggeration of all movements was carried out by a slight thrust with the index finger at the third cervical and counterforce by the operator's hand placed on the opposite side of the subject's head. The entire cervical procedure was repeated on the opposite side. The control made all movements carried out by the subject, but no mobilizing force was applied.

Immediately after the completion of all mobilization, the blood pressures and pulse rates were determined. The subject and control then rested in the supine position for a period of five minutes when blood pressures and pulse rates were again determined. This concluded the experiment.

Suboccipital Pressure

One hundred male students were used for this experiment to observe the effect of suboccipital pressure on blood pressure and pulse rate, fifty being used as subjects for manipulation and fifty as controls. The average age of both the subjects and controls was 22.2 years.

Subject and control assumed the supine position and rested for five minutes, at the end of which time the systolic and diastolic blood pressures and pulse rates were taken. In the subjects the following procedure was carried out: Bilateral suboccipital pressure was made by the operator's fingers, care being exercised that minimum cervical flexion, extension, rotation, side-bending, or traction was induced. This pressure was maintained for a period of two minutes, following which blood pressures and pulse rates were determined. Immediately after these readings, suboccipital pressure was again applied for two minutes, at the end of which time readings were again made to be followed in turn by another pressure period followed by readings of blood pressure and pulse rate. The subjects and controls were then allowed to rest in the supine position for five minutes, at the end of which time readings were again made, this time
without preliminary suboccipital pressure. All conditions of the experiment were repeated in the control group with the exception of the suboccipital pressure.

**Upper Thoracic Pressure**

One hundred male students were used for the experiment, fifty being manipulated subjects and fifty acting as controls. The average age of the subjects was 22.9 years, and of the controls 22.7 years.

Subject and control assumed the left lateral position and rested for five minutes. At the end of this time systolic and diastolic blood pressures and pulse rates were taken. In the subjects the following procedure was carried out: Bilateral upper thoracic pressure was made by the operator’s hands at the level of the upper three or four thoracic vertebral segments. Care was exercised that minimum flexion, extension, rotation, side-bending, or traction was induced. This pressure was maintained for a period of three minutes. Blood pressures and pulse rates were determined immediately after release of pressure. Subjects and controls then rested for five minutes, after which readings were again taken. All readings in the control group were made at the same time as in the subject.

**Cervical Soft Tissue Manipulation**

Sixty male students were used, thirty acting as subjects for manipulation and thirty constituting controls. The average age of the subjects was 23.3 years and of the controls 23.8 years.

Subject and control assumed the supine position and rested in this position for ten minutes. At the end of this time, systolic and diastolic blood pressures, and pulse rates were determined. In the manipulated subject, the following procedure was carried out immediately after the original readings were made: The operator stood at one side of the table and manipulated the posterior cervical soft tissues of the opposite side. Manipulation consisted of repeated stretching of the cervical muscles by forces exerted at right angles to their direction and without the application of traction or excessive rotation, these latter being controlled by the operator’s other hand. The fingers of the operator’s manipulating hand started at a point just lateral to the spinous processes and worked laterally around the posterior cervical region. The extent of the area covered was from the suboccipital region to the level of the seventh cervical vertebra. Manipulation was carried out in this manner for two and one-half minutes. Immediately upon conclusion of the manipulation, blood pressures and pulse rates were determined in subjects and controls. After a rest period of five minutes, blood pressures and pulse rates were again determined. This concluded the experiment.
Cervical Soft Tissue Manipulation Combined with Cervical Spinal Mobilization

Sixty male students were used, thirty as subjects and thirty as controls. The average age of the subjects was 21.8 years and of the controls 23.4 years.

Subject and control rested in the supine position for ten minutes. At the end of this period, systolic and diastolic blood pressures and pulse rates were determined. Immediately thereafter, the following procedure was carried out with the subject: The operator stood at one side of the table and manipulated the posterior cervical soft tissues of the opposite side. Manipulation consisted of repeated stretching of the cervical muscles by forces exerted at right angles to their direction. The force was applied by the operator’s fingers starting just lateral to the spinous processes and working laterally around the posterior cervical region extending from the suboccipital region to the level of the seventh cervical spinous process. This manipulation was continued for two and one-half minutes, and then the opposite side was manipulated in a similar manner for two and one-half minutes. The cervical spinal joints were then mobilized by the following procedure: A fixed point was established at the level of the third cervical vertebra. Rotation of the head and cervical column was carried away from the side of fixation, combined with slight extension and side-bending toward the fixed side. Rotation was carried to its limit; and when this point was reached, exaggeration of all movements was carried out by a slight thrust with the index finger at the third cervical vertebra and counter-force by the operator’s hand placed on the opposite side of the subject’s head. The procedure was repeated on the opposite side, both sides being mobilized within a few seconds following the soft tissue manipulation. Systolic and diastolic blood pressures and pulse rates were determined in subject and control immediately, and again after a rest period of five minutes. This concluded the experiment.

The results of these experiments are shown in figures 1, 2, and 3, which record graphically the changes in systolic and diastolic blood pressures and in pulse rates which occurred immediately after the manipulative procedure and after a period of rest following this procedure. The mean of changes for each group is shown, and also the maximum increases and decreases which were evident in both subjects and controls. None of the mean values shown has statistical significance. The maximum changes indicate a generally equal distribution between increases and decreases in both subjects and controls in all three figures.

These experiments would seem to indicate that the manipulative techniques used have little effect upon the blood pressure and pulse rate in a group of non-clinical subjects within this age range. These data may prove of value as frames of reference for future clinical investigations.
Fig. 1. The mean and extreme values for changes in systolic blood pressure induced by manipulation. First reading immediately after manipulation, second reading at interval after manipulation of 3 minutes in A and B, and of 5 minutes in all others. A—cervical mobilization; B—upper thoracic mobilization; C—lower thoracic and lumbar mobilization; D—mobilization entire spine; E—suboccipital pressure; F—upper thoracic pressure; G—cervical soft tissue manipulation; H—cervical soft tissue manipulation combined with cervical mobilization.
Fig. 2. The mean and extreme values for changes in diastolic blood pressure induced by manipulation. First reading immediately after manipulation, second reading at interval after manipulation of 3 minutes in A and B, and of 5 minutes in all others. A—cervical mobilization; B—upper thoracic mobilization; C—lower thoracic and lumbar mobilization; D—mobilization entire spine; E—suboccipital pressure; F—upper thoracic pressure; G—cervical soft tissue manipulation; H—cervical soft tissue manipulation combined with cervical mobilization.
Fig. 3. The mean and extreme values for changes in pulse rate induced by manipulation. First reading immediately after manipulation, second reading at interval after manipulation of 3 minutes in A and B, and of 5 minutes in all others. A—cervical mobilization; B—upper thoracic mobilization; C—lower thoracic and lumbar mobilization; D—mobilization entire spine; E—suboccipital pressure; F—upper thoracic pressure; G—cervical soft tissue manipulation; H—cervical soft tissue manipulation combined with cervical mobilization.
Summary

Results of the effects of eight types of osteopathic manipulation on blood pressure and pulse rate in a group of non-clinical college students are reported.

No statistically significant changes in systolic or diastolic blood pressure, or in pulse rate were obtained.

References

The purpose of this paper is to consider the clinical and experimental justification for the use of spinal tissue manipulation in rational therapeutic management of visceral disease. Before doing so, however, it will be well to review the frame within which the consideration will be held. This frame was established in two previous papers on the background and principles of rational therapy, and only a brief recapitulation of significant points made there will be set down here.

In this discussion the term "rational" has been taken to mean agreeing with observed events empirically determined. It may serve to focus this statement more sharply on therapeutic procedures if it is made to read agreeing with observed biological processes empirically determined. The ensuing discussion will include not merely the undisputed clinical fact that a given patient, or even patients in general receiving manipulative treatment regain health. It will include also experimental results which throw light on the physiology which must necessarily be involved if manipulative procedures are in fact responsible for the changes noted in visceral function. As will be seen in the development of the discussion there are serious gaps in the experimental evidence, and assumptions must be made. But clearly to understand just where these gaps in knowledge exist will direct attention to the need for further experimentation.

In this discussion the human organism is considered a physiological unit subject at all times from conception to death to a multiplicity of stimuli arising in both the internal and external environments of the body. The external environment, identical with that which surrounds inanimate objects, is the world in which all living organisms carry on their existence. It is at once the provider of the means of existence and the source of adverse forces which threaten that existence. It is for this reason that the relationship between the living organism and its environment is paramount in determining the relative success or failure of the organism in carrying on its biological functions of growth, maintenance, and reproduction. Because the demands upon the organism are in a state of perpetual change, the reactions of the organism cannot be static, but must shift to meet the demands. Yet the ultimate biological unit, the living cell, requires
an environment which is stable within quite definite limits if it is not to die. The environment with which the cell carries on its metabolic exchange is the totality of the circulating fluids of the body external to and bathing the cell. This is the internal environment of the body whose constancy is essential to the function of the cell, hence the body as a whole. This necessary condition of relative constancy is denoted homeostasis, and the means of preserving homeostasis are the homeostatic mechanisms. These are the mechanisms which mediate the adaptive changes in the internal environment elicited by both beneficial and harmful stimuli. The unhampered function of these mechanisms is essential to the preservation of a satisfactory relationship between the organism and the world in which it lives.

If it be accepted that health and disease are terms which denote respectively the degree of success or failure of the body as a whole in maintaining appropriate, integrated adjustments in reacting to environmental stimuli, and that in disease the integrity and adequacy of these mechanisms is compromised or overwhelmed by adverse forces whatever their source or kind, then it follows that therapy to be rational must be directed to the re-establishment of a relationship between patient and environment such that his reactions to stimuli shall fall within the physiological range which observation has shown constitutes homeostasis. It is on this general principle that any therapeutic method should be based, and it is by this general principle that every therapeutic method whether physical, pharmacological, or psychiatric should be judged as to its rational aspects.

This paper deals with one therapeutic method—manipulation of vertebral and paravertebral structures by procedures which are mechanical and designed to correct bad spinal mechanics. Their right to be included in any rational therapeutic plan may be judged by whether or not bad spinal mechanics constitutes an adverse force which compromises or overwhelms the integrity and adequacy of the homeostatic mechanisms, and whether or not manipulation by restoring good spinal mechanics helps restore homeostasis. The discussion will be confined to spinal mechanics without reference to the causes of faulty mechanics, and to the relation between spinal mechanics and visceral function. From this discussion a conclusion will be drawn as to the basis for manipulative therapy in a rational therapeutic plan. The evidence considered falls into two categories—clinical, based on experience in the correction of bad spinal mechanics, and experimental, based on laboratory procedures designed to elucidate nervous physiology. Both will be discussed.

*Spinal Mechanics*

It is a truism that the human body is mechanical to an important degree. Considered as a machine for locomotion and the performance of physical work the human body is made up of a complex but orderly
arrangement of bony levers held in apposition by ligaments, moved and held under control by striated muscles reaction to stimuli of the somatic nervous system. Considered as an energy transformer for the metabolism of ingested food the human body is a complex but orderly system of organs and glands reacting to stimuli of the autonomic nervous system. Cannon and others have shown that every expenditure of energy by striated muscle requires adjustment of smooth muscle and glands comprising visceral activity.

"Body mechanics," according to the report of the subcommittee on orthopedics and body mechanics of the White House Conference on Child Health and Protection published in 1932, "may be defined as the mechanical correlation of the various systems of the body with special reference to the skeletal, muscular and visceral systems and their neurological associations. Normal body mechanics," the subcommittee adds, "may be said to obtain when this mechanical correlation is most favorable to the function of these systems." In other words, "normal body mechanics" and homeostasis go hand in hand. In this report the body mechanics referred to—which affects to greater or less degree visceral malposition, reduction in the excursion of lungs and diaphragm thus interfering with metabolism, postural strain on antigravity muscles leading to fatigue, and strain on joint surfaces leading to arthritis—includes spinal mechanics as an integral part of overall body mechanics. The effect of gross deviation from good body mechanics on general health, its association with many visceral disturbances, and the concomitant improvement in health associated with the re-establishment of good overall body mechanics have long been recognized, so that this phase of the subject need not be elaborated here. Nor will consideration be given to direct injury to nerves in the intervertebral foramina by disc herniation, tumor, fracture, or dislocation. Instead, attention will be directed to the mechanics of the spine, to what constitutes good spinal mechanics, and to the more elusive mechanical derangements which occur in the spine and paravertebral tissues which will be designated alterations in spinal mechanics, or more briefly—altered mechanics.

Mechanics implies motion. Motion implies work performed. Work in this instance is the resultant of the force of muscle contraction applied to vertebrae, the parts moved. In spinal mechanics—as in any machine—the size, shape, and position of each functioning part, the motive force employed and its application to the moveable parts, in short the design of the assembly determines the force which can be applied to produce motion, and the pathway through which motion takes place. Good spinal joint mechanics obtains when all the moving and supporting parts of the spinal joint assembly perform the functions for which they are designed, with the least expenditure of energy. The conditions necessary to this include the postural requirement that the bodies of the vertebrae transmit the main compressive forces, that the facets, which are designed to stabilize and to guide vertebral motion move as the result of smooth co-
ordinated muscle action in pathways adapted to their shape, and that all spinal ligaments, including the intervertebral disc, permit this type of motion. In addition to these kinetic criteria, optimal spinal mechanics requires that spinal joint motion shall start from such a position that there is always a reserve of anatomically possible motion over and above the range habitually employed. The importance of this factor of safety motion to good spinal mechanics resides in the fact that motion to the extreme limit imposed by the anatomy of a joint introduces the possibility of strain or trauma.

If this concept of good spinal mechanics be accepted, it follows that bad spinal mechanics results from any significant failure to satisfy the criteria of good spinal mechanics. It will be noted that these criteria are based on three fundamental factors: The shape and position of the vertebrae, the shape of the pathway through which the vertebrae move, and the coordinated function of the muscles and ligaments. Alteration in any one of these factors inevitably entails alteration in the mechanics of the spinal joint assembly. The term “osteopathic spinal lesion” has been applied to this general situation, but there have been so many definitions of the term offered, each varying in some degree from all the others, that it seems better to consider the elements entering into altered spinal mechanics which are discoverable by diagnostic methods presently available to the practicing physician. The different factors will be considered as conditions which contribute to altered mechanics interfering with the optimal function of a spinal joint assembly.

The function of muscles attached to the vertebrae is either to maintain postural relationships or to produce spinal joint motion. Unusual increase or decrease in muscle tonus, even though bilaterally symmetrical, contributes to altered mechanics through changing the relative position of adjacent vertebrae and altering facet relationships. In the areas affected this at best reduces the factor of safety motion, and may put unusual strain on joint surfaces and joint capsules. If the muscle action be bilaterally asymmetrical, not only is static strain introduced by altered vertebral position, but in addition vertebral motion tends to be forced out of the path for which the angulation and shape of the facets are adapted, again putting unusual strain on facet surfaces and joint capsules.

The amount of motion permitted in a joint depends in part on how tight or loose the ligaments are. Injury to a ligament on one side of a joint which results in fibrotic changes and shortening alters both the range and character of motion possible in that joint. The joint is thereby forced to work at a mechanical disadvantage and, because the position of vertebrae above and below must be shifted to maintain the body in equilibrium with gravity, their factors of safety motion are reduced, increasing the potential likelihood of damage.

This discussion, deliberately abbreviated and held to simple terms, is sufficient to make clear that it is not necessary to predicate either develop-
mental anomalies or gross deviations from good body mechanics for a state of altered mechanics to exist in the spine and paravertebral structures. The evidence that the state of somatic spinal tissues designated altered mechanics affects the functional level of visceral organs will now be considered.

The Relation of Spinal Mechanics to Visceral Function

It is observed in clinical experience that with the variation in visceral function which exceeds the physiological range consistent with health there often co-exists a state of altered spinal mechanics clearly recognizable by present diagnostic methods. It is further observed that restoration of good spinal mechanics by manipulative procedures is in most cases followed by clinical improvement in the patient. An isolated instance of this sequence of events would be interesting but not significant. But the cumulative clinical evidence that a change from bad to good health consistently accompanies the change from bad to good spinal mechanics becomes impressive when manipulative procedures are the only therapeutic method employed. On the clinical evidence alone manipulation must be, and is, given credit for being a potent therapeutic aid to the patient's improvement. But to explain these observed events it is necessary to establish how the mechanical status of vertebral and paravertebral somatic structures exercises control over visceral function.

Physiologists have amassed ample experimental evidence of the integrated action of somatic and autonomic nervous systems, and of the anatomical representation of the autonomic nervous system at all levels of the neuraxis. The situation may be summed up by saying that the experimental demonstration of this correlation validates the doctrine of homeostasis, that the autonomic nervous system is a most important part of the homeostatic mechanisms which preserve the delicate balance of the internal environment necessary to health, that somato-visceral reflexes form the basis for autonomic reactions which adjust the internal to the external environment and to every action of the body in which striated muscle takes part. There is also a growing volume of evidence regarding the organization of the autonomic nervous system into successively higher and more reactive levels of integration. These levels begin with the peripheral ganglia which seem able to maintain a basic tone in the sympathetic and parasympathetic systems even when cut off from the central nervous system. In the intact animal this basic tone is modified by segmental centers in the spinal cord, and by suprasegmental centers in the medulla which contains the principal centers regulating vasomotion, adrenin secretion, and respiration, in the hypothalamus which seems to be the highest subcortical center completely co-ordinating somatic and autonomic reactivity, and in the cortex which acts on lower centers and where the extensive autonomic representation brings about the more or less discrete functional integration exemplified by the instant correlation between heart
rate and localized somatic muscular activity. In adjusting the internal environment these integrating centers react to afferent impulses set up by exteroceptors stimulated by changes in the external environment, and to afferent impulses set up by receptors in muscles responding to the dynamics of the body in relation to gravity. What is not known from experiments is either the physiological laws operating between the posterior horn cells of termination and the visceral efferent nuclei in the lateral cell column, or the exact nervous pathways which transfer somatic afferent impulses to autonomic efferent neurones. It is this lack of experimental evidence which necessitates the postulation of a theory to explain how somatic vertebral and paravertebral structures exercise a regulating influence over visceral function.

The theory offered is based on the concept that control of visceral function is a part of general homeostatic control effected by the neurohumoral system. For simplicity in presentation only the neuronal component will be discussed here. This is deemed admissible because any effect manipulative procedures applied to paravertebral tissues may have on endocrine activity is in all probability mediated over the nervous system. It will further simplify the discussion to limit consideration to the sympathetic division of the autonomic nervous system. This too is admissible in the development of a possible explanation because while both sympathetic and parasympathetic divisions are known to respond coincidentally in some, and probably do so in all important adjustment reactions, the sympathetic has greater reactivity and is recognized as dominant in effecting adjustment.

The nervous pathways over which vertebral and paravertebral structures might exercise control over visceral function by causing alterations in sympathetic activity are the local segmental reflex arcs and those involving the higher integration centers in the central nervous system, notably those in the medullary and hypothalamic nuclei. By whatever pathways the nervous control is exercised the afferent limb entering the central nervous system over posterior spinal roots is made up of somatic sensory nerves with receptors in the skin and subdermal structures, the paravertebral muscles, and the articular and periarticular structures. The efferent limb leaves the central nervous system over anterior spinal roots and is made up of autonomic neurones whose central origin consists of preganglionic neurone cell bodies grouped in the visceral efferent nuclei of the lateral cell column. These preganglionic sympathetic neurones, with the exception of those supplying the adrenal medulla, pass to one or more peripheral ganglia where synaptic junctions are made with postganglionic neurones proceeding to an effector organ, thus completing the reflex arc connecting vertebral and paravertebral structures with a visceral effector organ. This reflex arc provides the anatomical pathway over which physiological control may be exercised. The experiments of Bronk and others using the superior cervical ganglion of the sympathetic chain shed light on the
physiological laws which operate in effecting this control, and will be utilized in constructing the theory presented.

In general the response of an effector organ is graded by the frequency of the impulses reaching it. This frequency in turn varies with the frequency carried by each fiber, and with the number of fibers in action. In the case of effector organs innervated by the autonomic nervous system, the nerve fibers involved are postganglionic; and the activity of each postganglionic fiber is determined by the activity of its nerve cell located in a ganglion. The frequency with which a postganglionic neurone cell body discharges is determined by its antecedent activity which has an important effect on its irritability, by the combined effect of various environmental agencies such as the concentration of acetylcholine, potassium, calcium, and oxygen in the fluid bathing the nerve cell, and by the summation of impulses reaching the postganglionic nerve cell over several preganglionic fibers in accordance with the phenomenon of spatial summation. To this point it is seen experimental evidence demonstrates that the number and activity of preganglionic nerve fibers conducting impulses from visceral efferent nuclei in the central nervous system, and acting on postganglionic nerve cell bodies in peripheral ganglia are important in regulating the response of visceral effector organs.

Turning now to the afferent limb of the reflex arc involved in the possible reflex control of visceral function, it is known that the somatic structures comprising the spinal joint assembly are amply supplied with sensory receptors in the skin and subdermal structures, in the paravertebral muscles, and in the articular and ligamentous structures, and that there are proprioceptors in the muscles and tendons. It is well established in physiology that the intensity of a stimulus applied to a receptor grades the frequency of impulses transmitted by the afferent fibers to the central nervous system, that, specifically in muscles, muscle tone grades the stimuli originating in muscle nerve receptors, and that the phenomenon of spatial summation operates to activate a pool of secondary neurones in the posterior horn. To explain the clinical effect of paravertebral somatic status on visceral function it must be assumed that impulses reaching the posterior horn over dorsal spinal nerve roots activate internuncial neurones which either directly, or by relay through suprasegmental integration centers, terminate on the preganglionic autonomic efferent cell bodies grouped in the lateral cell column. Consideration must be given to both routes within the central nervous system because in the study of centers controlling the secretion of adrenin, reflex stimulation seemed to have slight effect on spinal centers. But whether one or both routes are utilized there are anatomical means provided whereby afferent stimuli originating in paravertebral somatic structures may reach visceral efferent nuclei containing preganglionic nerve cell bodies. It must be further assumed, in the theory proposed, that these centrally located autonomic nerve cell bodies are subject to the same classes of influences demonstrated as controlling
the activity of postganglionic cells located in peripheral ganglia. This combination of known and assumed anatomy and physiology provides an explanation of how the mechanical status of vertebral and paravertebral structures, by grading the frequency of impulses reaching a visceral effector organ, can exercise a regulating influence over visceral function.

The Relation of Spinal Manipulation to Visceral Function

If this theoretical explanation prove true to physiological laws it becomes possible to state that manipulation of vertebral and paravertebral structures can influence visceral function in at least two different ways. It can eliminate the cause of unusual stimuli in articular and periarticular structures, and in hypertonic paravertebral muscles by re-establishing the conditions requisite to good spinal joint mechanics. In other words, it becomes possible to state with reasonable assurance that the therapeutic value of spinal manipulation is due to re-establishing conditions in which unhampered functions of the neuronal homeostatic mechanisms can restore homeostasis. The second possibility, that manipulation, by introducing new stimuli, can produce direct stimulation or inhibition acting over post-ganglionic fibers cannot be ruled out, but is not necessary to a satisfactory explanation. If the theory offered be substantiated, there is an experimental as well as a clinical basis for including spinal manipulation in rational therapeutic plans for the management of visceral disease. It is not maintained that manipulation is all sufficient. But it is claimed that to the extent the physiology of the body is regulated by spinal mechanics, to that extent mechanical manipulative procedures directed to eradicating faulty spinal mechanics and reinstating structural relationships which co-exist with homeostasis, are indicated parts of any rational therapeutic plan. Merely to mention the present need for drugs in the management of malaria or syphilis, for surgery or radiation therapy in neoplastic diseases, for specific antitoxins in diphtheria, for dietary measures in pellagra or rickets, for endocrine therapy in hypothyroidism, for psychotherapy in the neuroses, is sufficient to render untenable any position which does not recognize the need for employing all feasible indicated methods provided by the several branches of medicine. The reason for advancing the theory offered in this paper is to point out the gaps in knowledge which call for experimental investigation.

Summary

The clinical and experimental justification for the use of spinal tissue manipulation in rational therapeutic management of visceral disease is discussed.

A theory to explain how spinal manipulation regulates visceral function is presented.

It is concluded that if this theory be substantiated there is an experimental as well as a clinical basis for including spinal manipulation in
rational therapeutic plans for the management of visceral disease.
The present necessity for all other rational therapeutic methods is
recognized.
The lack of conclusive proof of how spinal manipulation effects
changes in visceral function, and the need for experimental investigation
is emphasized.

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Mr. W. T., aged 54, a teacher, was admitted to the Urological service in the hospital on November 5, 1943. At that time he was suffering from a pain in the back, had a temperature of 100.2°, and had a palpable mass in the abdomen.

The patient stated that prior to September 15 he had not been aware of any abnormality. It was his custom to go to his physician each year for a complete physical examination, and at the last visit the tumor had been revealed. He stated that on finding the mass in his side, his physician had made various laboratory studies to determine the nature of the enlargement. These had included a gastro-intestinal x-ray study, which placed the lesion outside the abdominal cavity. Intravenous urography was attempted but dye concentration was practically nil, and none of the renal structures was visualized. Retrograde pyelography was then attempted in an effort to visualize the renal calyces. The patient reported that "the doctor was unable to get up the ureters." In spite of this, a diagnosis of polycystic disease was made. Cystoscopic examination was carried out on October 4, and immediately following this procedure the acute symptoms developed.

He had had measles, chicken pox, and pertussis, and stated that as an infant he had had "epileptic fits." His personal history was otherwise negative except that there was slight constipation, and he arose twice during the night to urinate. His mother had died of carcinoma. His family history was otherwise negative.

On admission to the hospital he appeared to be a desperately ill man. There was marked tenderness over the right renal area and the tumor. There was constant twitching in the hands, feet, and head. His facies and general appearance was that of the uremic state, although at the time of admission the uremic odor was not present.

Examination of the blood gave the following results: Hemoglobin, 52.3 per cent (8 gm. per cent); leucocytes, 20,000 per cu. mm.; lymphocytes, 4 per cent (800 per cu. mm.); polymorphonuclear leucocytes, 94 per cent (18,800 per cu. mm.); and transitionals, 2 per cent (400 per cu. mm.). The rate of blood sedimentation was 32 mm. in twenty minutes, and this increased to 36 mm. in forty-five minutes.
Analysis of the urine showed a specific gravity of 1.012, slight turbidity, an acid reaction of pH 5, and a positive reaction for albumin. The urine was otherwise essentially negative except for an occasional hyaline cast and one to two pus cells per H.P.F. The urine was negative for blood.

Survey x-ray films were made, and while this was being done the patient nearly died of a cerebral crisis while on the table. Wet film examination at this time was unsatisfactory because the patient was filled with gas, although it did reveal a large mass in the left flank which was reported to be suggestive of a "hydronephrosis, or an enlarged kidney. Visualization of the right kidney was not possible due to gas."

Diagnosis of cyst, infected hydrops, or tumor which had become cystic was made, and it was felt advisable to attempt drainage of the cavity which was visualized on the film. Accordingly, under local analgesia a small incision was made over the left flank, and the kidney was immediately found upon separating the muscles. It was definitely cystic in the area visualized. A small wound was made into the largest cyst, and a catheter was introduced into the cavity. A large amount of pus was immediately withdrawn and approximately a quart and a pint was drained ultimately. Blood transfusion was given, and the patient had an uneventful night. His temperature fell and he appeared much better the following morning. During the following day his temperature rose to 100.4° and on November 8 to 101.4°, but from then until the time of his death the temperature decreased progressively.

Blood urea nitrogen at this time was 127.4. The patient still twitched, although he was perfectly clear mentally and stated that he felt fine. On November 8, uremic frost was noted on the skin. He was in no pain, but was constantly bathed in perspiration. The twitching of his limbs was increased, and he was quite nervous about his condition. On November 10, his B.U.N. was 274, and on November 12 it was 287. On November 12, urinary incontinence developed, and he became definitely worse although he remained conscious. On the morning of November 13 he became slightly confused at times, although at other times he was perfectly rational. He expired at 11:55 p.m. that night. Immediately before death his B.U.N. was 290.

Permission for necropsy was granted.

Autopsy No. 43-312
Died: 11:55 p.m., 11-13-43
Autopsy: 4:00 p.m., 11-14-43

External Examination

The body was that of a pale, emaciated, middle-aged adult male estimated to weigh 145 pounds and measuring 67 inches in height. The entire body was in the height of rigor. The head was well formed but
supported only a thin diminishing crop of hair. The pupils were equal in size. A reasonable degree of oral hygiene had been maintained. Nothing noteworthy was noted in the cervical region. The thorax was fairly well formed with the exception of a shallow Harrison's sulcus. Abdominal distention was marked. An oblique incision in the left flank, 11 cm. long, of very recent origin was seen. Suture material had not been removed. The extremities were symmetrical and well shaped. Small hematomata was seen in both anticubital fossae. There were 3 mm. of adipose tissue in the midline.

**Internal Examination**

The pericardial cavity contained less than 25 cc. of yellowish fluid. It was immediately evident that the visceral layer of the pericardium was sandpaper-like in its texture. Very fine adhesions were found uniting the visceral layers between the atria and ventricles and the atria and the aorta. No anomalies of the great vessels were present. The pulmonary artery contained no emboli. The greatest diameter of the heart was 14 cm., that of the thorax at the upper dome of the diaphragm 20 cm., giving a cardio-thoracic ratio of 14/20. The dome of the diaphragm was displaced to a higher level than normal by the masses in the renal areas of the abdominal cavity.

The heart measured 14 x 12 x 7 cm. and weighed 350 grams. The evidence of acute pericarditis has been presented. The right atrium and ventricle were dilated, and the myocardium showing myomalacia. The valves were average in status. Numerous "chicken fat" clots filled the enlarged right heart. The myocardium of the left heart was also myo-

*Fig. 1—Photograph of right and left kidneys post-mortem.*
malacic. The posterior leaflet of the mitral curtain was slightly deformed and contained an atheromatous patch which was somewhat calcified. The aortic cusps were normal. There existed a patent foramen ovale which was 1 cm. in diameter. A generalized atherosclerosis of the coronaries was found. Most noteworthy was an atheromatous patch which occluded about three-quarters of the lumen of the anterior descending limb of the left coronary.

The aorta showed a blackish discolored atheromatous ulcer, 3 cm. above the bifurcation of the iliacs, about 1 cm. in diameter. Many atheromatous patches were scattered throughout the aorta.

The right and left pleural cavities were free of adhesions and fluid.

The right lung showed the presence of a calcified Ghon lesion in the middle lobe. Old tubercular invasion was also evidenced by the finding of enlarged calcified mediastinal lymph nodes. Bloody, frothy debris was found in the terminal bronchioles. Congestion of the bronchial tree was marked. There was abundant edema in the lungs. The left lung was slightly smaller than usual. Its apex lodged a reinfection lesion of tuberculosis. Aspirated vomitus was found in the congested bronchi. Hypostatic congestion was severe in the left lung but less edema was present than on the right side.

The peritoneum, when opened, allowed the escape of much gas. A small amount of bloody fluid was found in the peritoneal cavity. The omentum was thin and covered areas of devitalized bowel. Upon lifting it, two large masses were seen displacing the intestines from both renal areas but especially the left. Calcified tubercules of tuberculosis were noted in the mesentery of the terminal coils of the ileum. The appendix could not be found. Numerous adhesions were found about the gall-bladder and hepatic flexure of the colon.

The esophagus contained a very small amount of vomitus.

The stomach was somewhat dilated by fluid material and gas. The prepyloric region was somewhat deformed by a cicatrix, possibly the remnant of a healed ulcer. The duodenum and its ligament of Tritus was displaced to the right of the midline by the mass in the left renal area. It was impossible to demonstrate the foramen of Winslow. The small bowel was somewhat devitalized. Congestion was present in the terminal portion of the ileum. There were no evidences of tubercular ulceration. The ascending colon was somewhat devitalized. The hepatic flexure was bound to the liver by adhesions. The transverse colon was displaced with fecal matter and congestion. Its terminal portion and the descending colon were displaced to a position of compression between the mass in the left renal areas and the abdominal wall. This portion, thus impinged upon, presented a marked inflammatory reaction due to crushing, and a good deal of recent hemorrhage was found in and about it. Complete obstruc
POLYCYSTIC DISEASE OF THE KIDNEY

The liver was large, weighing 1780 grams. Its consistency was of such firmness as to indicate a slight degree of cirrhosis. The gallbladder was greatly distended with heavy red-brown bile, which could only be made to flow with difficulty from the extrahepatic biliary ducts. A few mulberry calculi with a fine precipitate of biliary pigments were contained in the gallbladder. There was no cholesterolosis.

The pancreas showed a normal lobular pattern.

The spleen measured 12 x 8 x 3 cm. and weighed 180 grams. A defect was noted in its capsule possible due to a tubercule. It was of a rusty color.

The right adrenal gland weighed less than five grams and was of average size and shape. It was somewhat edematous, but the medullary substance was well preserved. The left adrenal gland weighed less than five grams but was of distorted shape and crushed by the mass below.

The right kidney measured 23 x 12 x 9 cm. and weighed 1300 grams. (Figure 1.) It was polycystic, the cysts varying in size from the microscopic to 3 to 4 cm. in diameter. The capsule stripped with ease. Upon its removal, a purulent exudate oozed from the torn surface. Upon closer inspection the cysts were seen to have contents of three types. One was that of hemorrhage, another of the purulent exudate, and the third type contained clear yellow fluid. The left kidney measured 29 x 15 x 12.5 cm. and weighed 2500 grams. It was also polycystic, being made up of cysts of approximately the same size as the right with the exception of several cysts about 6 to 7 cm. in diameter. These cysts also contained the three types of fluid mentioned above. However, in this kidney those cysts containing purulent debris predominated. The capsule stripped with ease allowing much of the purulent, bloody exudate to flow out. The capsule had been entered as it lay adjacent to the left flank incision already mentioned. A moderate amount of hemorrhage had infiltrated surrounding tissue, and several large clots were removed from the perirenal capsule. The right ureter was of normal size and status. The left ureter was forced to travel slightly upward before it could pass posteriorly and downward behind the kidney since the latter had shifted to a position in which the lower pole lay at a point more to the right, more superiority, and more anteriorly than normal.

The bladder contained 75 cc. of turbid urine and some sediment. The ureteral orifices were patent and in normal position. The prostate was of such a small size as to make its identification doubtful.

The external genitals were smaller than average. Phimosis was present. Both testicles were present but were very small.
Ante-Mortem Blood Chemistry (immediately before death)

Blood urea nitrogen 290 mgm. per cent
Blood sugar 110 mgm. per cent

Post-Mortem Blood Chemistry

Blood urea nitrogen 333 mgm per cent
Blood sugar
left ventricle 98 mgm. per cent
right heart 132 mgm. per cent
portal vein 249 mgm. per cent

Anatomic Diagnosis

Acute pericarditis
Myomalacia
Cardiac dilatation
Coronary atherosclerosis and occlusion
Atherosclerosis and atheromatous ulceration of aorta
Pulmonary edema
Abdominal adhesions
Healed peptic ulcer
Colitis (descending colon)
Cirrhosis of the liver
Cholelithiasis
Polycystic kidneys with superimposed infection
Phimosis

Cause of Death

Immediate—Polycystic disease of the kidneys (infected)
Contributory—Myomalacia and uremia.

Discussion

Polycystic disease of the kidney is a congenital bilateral disease, and this patient lived beyond the average age for this condition. He had remained symptomless until shortly before his death, although undoubtedly there had been palpable kidneys for some months or years prior to this. A properly conducted physical examination should have discovered the lesions before the time that they were finally diagnosed. Polycystic kidneys are easily infected, and in this patient the slight trauma produced at cystoscopy was the exciting cause of his pyonephrosis. Death in these cases results either from renal failure with uremia, or from sepsis fol-
lowing infection. In this case both factors were present. There was almost total destruction of renal structure as regards functioning tissue, and this case illustrates how a patient may feel in apparently good health with almost no renal tissue.

**Summary**

A case of polycystic disease of the kidney in a male, age 54, is presented. The patient had remained relatively symptom-free until near the time of his death.

Autopsy findings are reported.