ON BREATH SOUNDS

by

A. R. NATHOO

A Thesis Presented for the Degree of Doctor of Medicine in the

University of London

September, 1972.

ABSTRACT

The subject of this thesis is breath sounds in health and disease. It is an investigation of the nature and site of production of normal breath sounds, and of their variations in chronic bronchitis, asthma, primary emphysema and bronchial and tracheal stenosis.

The breath sounds were recorded at the mouth during inspiration, and their intensity was correlated with the instantaneous volume flow rate. At a similar flow rate, the intensity of inspiratory breath sounds in patients with diffuse airway obstruction due to bronchitis or to asthma was greater than in normal subjects. In these patients the inspiratory sound intensity correlated inversely with the maximum mid-inspiratory flow rate, the forced expiratory volume in one second and the peak expiratory flow rate. In patients with primary emphysema, however, the inspiratory sound intensity was normal in the presence of severe expiratory obstruction, and in patients with bronchial and tracheal stenosis the inspiratory sound intensity was greater than predicted from these forced expiratory tests.

The breath sounds are acoustically a white noise. It is suggested that they are generated by oscillations of gas within those airways where the airflow is non-laminar. These include the upper respiratory tract, trachea and the proximal intrathoracic airways. The intensity of breath sounds is flow rate dependent. When the airways are narrowed the linear velocity of airflow increases and more sound is produced. The intensity of inspiratory breath sounds in chronic bronchitis and asthma, it is inferred, reflects calibre changes of the trachea and the proximal bronchi.

Exercise and the inhalation of tobacco smoke increased the inspiratory sound intensity in some asthmatic subjects. Bronchodilator drugs reduced the intensity of inspiratory breath sounds in chronic

bronchitis and asthma when it was increased. The changes in inspiratory sound intensity after bronchodilator drugs was of similar magnitude to the changes in the spirometric tests of airways obstruction.

The breath sounds recorded on the chest wall contain a lower range of frequencies than the breath sounds at the mouth. It is suggested from the evidence presented that these sounds do not originate in the terminal bronchioles and the alveoli, but are transmitted from proximal bronchi.

-- To Paul Forgacs --

CONTENTS

LIST OF TABLES AND FIGURES

I_s = Inspiratory sound intensity, PEF = Expiratory flow rate, FEV_1 = Forced expiratory volume in one second, VC = Vital capacity, R_{aw} = Airway resistance, SR_{aw} = Specific airway resistance, MMIF = Maximum mid-inspiratory flow rate.

Fig 1

A 19th. century drawing from F.V. Mérat's article (1819) entitled "Pectoriloque". This article includes a plate of the stethoscope reproduced from Laennec's book with the addition of this drawing which shows the stethoscope in use. This is probably the earliest illustration of mediate auscultation (Bishop, P.J. personal communication, 1972).

HISTORY

The Hippocratic writings contain some of the earliest references to auscultation. These include descriptions of the tracheal rattle, pleural rub and crepitations. Caelius Aurelianus in the 5th century A.D. wrote that in acute asthma "..... as the disease begins to grow more intense, there occurs wheezing and hissing sounds in the chest .....". None of the ancient writers seem to have referred to breath sounds or to systematic examination of the chest. The usefulness of this was perhaps first anticipated by Robert Hooke (1635 - 1703) : "... who knows, I say, but that it may be possible to discover the Motions of the Internal Parts of Bodies, whether Animal, Vegetable or Mineral, by the sound they make, that one may discover the Works perform'd in the several Offices and Shops of a Man's Body, and thereby discover what Instrument or Engine is out of order, ... And somewhat more of Incouragement I have also from Experience, that I have been able to hear very plainly the beating of a Man's Heart, and 'Tis common to hear the motion of Wind to and fro in the Guts, and other small Vessels, the stopping of the Lungs is easily discover'd by the Wheezing, the Stopping of the Head, by the humming and whistling Noises, the sliping to and fro of the Joynts in many cases, by crackling, and the like ...."

It was not until the start of the 19th century that regular auscultation of the chest was first developed. In 1808, Corvisart translated Auenbrugger's monograph "Inventum Novum" and added his own observations on percussion of the chest. Amongst his many distinguished pupils, Bayle and Laennec were the first to practise immediate auscultation of the heart. Laennec invented the stethoscope in 1816. Three years later, he published "De L'auscultation mediate" in which he described his stethoscope and his correlation of auscultatory signs with anatomical lesions. This work placed auscultation on a firm scientific foundation.

Laennec's examination of his patients was most searching. Even after the discovery of the stethoscope, he did not disregard the importance of immediate auscultation and routinely employed inspection, palpation and percussion in examination of the chest. He described lung sounds, classified them and indicated their significance.

Laennec distinguished two varieties of normal breath sounds which he referred to as "vesicular respiration" and "bronchial respiration". He described these sounds as follows:

> " ....If this form of stethoscope is placed against the chest of a man in good health, there will be heard, during both inspiration and expiration, a slight but most distinct murmur, which indicates the entrance of air into the pulmonary tissue and its expulsion. This murmur may be compared to the sound produced by a pair of bellows, the valve of which is noiseless, or better still, to that heard by the unaided ear when a man, in deep and peaceful sleep, periodically draws a long inspiration. It is almost equally audible at all points of the chest, but especially at those spots where the lungs are nearest to the cutaneous surface - that is to say, in the anterior, superior, lateral, and lower posterior regions. The hollow of the armpit and the space between the clavicle and upper edge of the trapezius muscle are the points where it is heard with greatest intensity.
>
> " We can hear it equally well over the larynx and over the bare or cervical portion of the trachea - and even in many men over the whole length of the windpipe down to the base of the sternum; but over the trachea, and also in some degree at the roots of the bronchi, the respiratory sound has a special

character, clearly showing that the air is passing through a roomier channel than the air cells; further, it often appears as if the patient, during inspiration, were drawing in the air contained in the tube of the stethoscope, again expelling it during expiration."

Laennec recognised that the intensity of breath sounds varied with the rate and depth of respiration, and that the breathing of children was much louder than that of adults. One of the important observations he made was on the breathing of an asthmatic subject examined without the aid of the stethoscope. He wrote:

"The respiration which to the unaided ear appears loudest is not necessarily that which makes the greatest noise inside the chest. I do not here refer to breathing accompanied by rale, whistling or any other foreign noise, but to respiration, which is only loud and which occurs in many acute and chronic maladies, and particularly in persons suffering from dyspnoea, no matter what the cause. This noise, which is, as it were, merely the exaggeration of that produced by many men when breathing during sleep, and which can be easily imitated at will, arises entirely in the nasal fossae and at the back of the mouth, and is occasioned solely by the manner in which the air striked the edge of the glottis, the soft palate, and the walls of the nasal fossae. I am acquainted with a man, suffering from asthma following upon dilation of the ventricles of the heart, whose respiration can ordinarily be heard at a distance of twenty paces. The murmur produced inside the chest by inspiration and expiration is in his case less strong than in the majority of men.

The same is true also of the snoring of a healthy man during sleep.

To complete this series of observations, I thought it might be useful to examine the chest of one of those mountebanks who with their voice imitate perfectly the sounds made by a saw, plane, &c., &c. I obtained the same result: the noises arise in the back of the mouth and the nasal fossae; they are due to the manner in which the inspired and expired air vibrates in those parts, and are without any influence on the respiratory murmur."

Laennec's work on auscultation soon received wide recognition. He found able support from Piorry, Bouillaud, Bertin and Andral in France; Skoda in Austria, Schönlein and Krukenberg in Germany; Forbes, Hodgkin, Hope, Elliotson and Williams in England; Stokes, Graves and Corrigan in Ireland and Jackson, Gerhard and Flint in America. These physicians among others, greatly extended his observations and even described new sounds.

The breathing of asthmatic subjects attracted Andral's attention. Describing a patient with acute asthma, he observed:

" .... there is heard in all points of the chest a general hissing sound (sifflement) which accompanies each inspiration, and which in certain cases is also heard in the larynx. In this latter organ, as in the large and small bronchi, the sudden appearance of the hissing respiration can be explained in no other way but by the equally sudden engorgement of the laryngo-bronchial mucous membrane; thence the fit of asthma. ...."

(Andral, 1836).

Ramadge (1847) interested in the breathing of asthmatics, clearly distinguished the sound of wheezing from a "hiss". He wrote:

> "The character of the respiratory sound in asthmatic cases varies from a variety of circumstances. Thus the "rale sonore", the sonorous rale, so far from presenting one uniform character, is divided into several sounds perfectly distinct from each other. At times, it resembles the sighing of the wind through the trees; at others, that of air violently forced through a tube, as is the case with the bellows of a forge; and at others again, it acquires a sharper sound, something between a hiss and a whistle. ...."

The observations on loud breathing and "hissing respiration" of asthmatic patients aroused little interest in the 19th century. Instead a great controversy followed as to the origin of normal breath sounds.

Laennec did not specifically state the site and the mode of origin of normal breath sounds. It was usually inferred from his writings that he believed breath sounds resulted from the friction of air on inner surfaces of the trachea, bronchi and the pulmonary vesicles during inspiration and expiration. This view was generally accepted though not based on any experimental evidence. Skoda (1853) wrote:

> "I explain the sound of the vesicular breathing as Laennec does, attributing it to the friction of the air against the walls of the finer bronchial tubes and the air-cells, the contractile power of which it has to overcome. The reason why the inspiratory murmur of the air-cells is much louder than the expiratory is, that the air, when it enters into them, meets with resistance from their contractility, but does not meet with any in its passage out of them. It is otherwise, however, with the large bronchial tubes, and particularly with the trachea and larynx; here the air, during inspiration, meets with no opposition - it has, indeed, rather a tendency to expansion;

> but during expiration, being forced from a larger - the air-cells - into a smaller space, it is compressed; and hence the expiratory murmur of the larynx, the trachea, and the largé bronchi, is, as a rule, louder than inspiratory."

Beau, a contemporary French physiologist, proposed in 1834 an alternative explanation of the mode of production of breath sounds. He believed that all normal breath sounds originated from a single source in the posterior part of the mouth, mainly the velum palati. This sound resulted from friction of inspired and expired air at this site and he termed this "bruit guttural". The sound heard through the medium of the stethoscope was the same sound but modified during transmission. The observation which originally led him to doubt the conventional view was made on a patient with pleural effusion. This patient exhibited bronchial breathing accompanies by loud breathing at the mouth. He found that the intensity of bronchial breathing could be diminished or abolished by making the patient diminish or suspend the respiratory sound in the throat. Beau's theory received support from Spittall (1839) and Gee (1877). Bushnell (1921) attempted to demonstrate that the vesicular breath sounds originated in the larynx and later believed that they resulted from vibration of the vocal cords (Pratt and Bushnell, 1925).

Beau's work met with strong criticism from William Stokes (1837) and C.J.B. Williams (1840), a distinguished English pupil of Laennec. Stokes wrote:

> "I have carefully repeated the experiments of M. Beau, and feel convinced that his conclusions are erroneous, ...
>
> I shall here allude to a single fact, which, in itself is sufficient to overturn the theory; it is that we can hear a natural respiratory murmur in patients who do not breathe through the mouth or nostrils. Of this we can easily satisfy

> ourselves by examining a person who has been operated on for laryngeal obstruction, and who breathes through a fistula in the trachea. I have now examined eight of such cases, and found that in all the respiratory murmur could be heard with ease ...."

Williams (1840) argued that the voice sound was not uniformly transmitted in all parts of the chest and observed that when the vesicular sound was loud, the bronchial component was not correspondingly exaggerated. He also noted that when laryngeal rhonchus supplanted laryngeal blowing, the vesicular murmur still persisted in addition to the transmitted rhonchus.

Stokes and Williams among others, favoured Laennec's explanation of the origin of the normal breath sounds. During inspiration and expiration the bronchial breath sounds were generated by air friction in the trachea and at all levels in the bronchial tree, and the vesicular breath sounds were produced in the air vesicles. The sound in the superior air passages played no part in the genesis of normal breath sounds. However, the expiratory component of the vesicular murmur could not be explained on this hypothesis for it was recognised that this sound varied from the inspiratory sound not only in duration but also in character in which it resembled the bronchial sound. This difficulty led to the conclusion that the expiratory part of the vesicular murmur did not originate in the alveoli but was transmitted from proximal bronchi.

Walshe (1871), commenting on the origin of vesicular breath sounds, wrote:

> " .... and it will probably be admitted by any impartial person that in the present state of thoracic physics no single view hitherto tendered is capable of being demonstrated. Fortunately this unsettled state of the physiological question

throws no cognizable difficulty in the way of practically interpreting the signification of alterations from the types of health."

In the late 19th century, experimental evidence was sought to explain how and where breath sounds were produced. The theory of "fluid vein" enjoyed a temporary popularity. According to this theory, a blowing sound was generated when fluid (liquid or gas) passed from a narrower to a wider space. Hence, during inspiration, sound was produced at two sources; the glottis and at points where the bronchioles opened into the pulmonary vesicles. During expiration, sound was generated at the glottis only. The experiments of Bondet and Chauveau (1887) were thought to support this view. They submitted a horse suffering from lobar pneumonia to a tracheostomy thus abolishing the conditions for "fluid vein" at the glottis. With the tracheostomy wound held open, they observed that the inspiratory bronchial sound over the pneumonic lobe disappeared whereas the vesicular sound over the rest of the lung persisted. Bondet divided the 'pneumogastric' nerves (vagi) of another horse and found that the vesicular murmur was abolished. He believed that when the pneumogastric nerves were divided, the bronchiolar walls were paralysed so that "fluid vein" no longer existed at the alveolar level (Brit. For. Med. Chir. Review, 1873).

Sahli (1872) described a case of pulmonary hernia due to sternal fissure. He found that when the patient was instructed to perform a valsalva manoevre thereby eliminating laryngeal or glottic sounds, the vesicular murmur could still be heard. He inferred that at least a part of the vesicular sound must have been locally generated.

Bullar (1884) devised a series if experiments to determine in what part of the respiratory tract the breath sounds were produced. He used a pair of sheep and calf lungs excised at a level just above the

bifurcation of the trachea. The left lung was placed in an airtight water chamber simulating the thorax, the right lung rested on top of the chamber in a collapsed state. The left lung could be made to inflate or deflate by applying negative or positive pressure around it. When the left lung was inflated the sound heard over this lung was vesicular in quality, whereas over the right lung it was bronchial in character. The trachea was then plugged so that deflating the left lung caused the right lung to inflate. A vesicular sound was now heard over the right lung. He also found that a deflated lung conducted sound better than an inflated one, and that a new sound was produced at the site where a bronchus was narrowed. His main conclusion on the origin of vesicular sound was as follows:

> "... The only tenable conclusion of these facts is that the vesicular murmur is produced in the lung itself; for if it be not produced in the lung it must be a conducted sound, and we have just seen that expansion of the lung diminishes its conducting power; but the peculiarity of the vesicular sound is that it is heard loudest during inspiration, that is to say while the lung is expanding, and by so doing becoming a worse conductor of sound. The sound is loudest when the conducting power of the lung is least; it cannot, therefore be a conducted sound."

Even though most physicians of the 19th century speculated on the possible mechanism and site of production of breath sounds only a few investigators, already quoted, attempted to determine this by experimental evidence. There were two main reasons for this. In the first place, to a practical physician it was sufficient that a particular auscultatory sign and the disease, established by pathological examination, should go together. Flint (1856) expressed this view in the following way:

> ".... As already remarked, knowledge of physical signs, their significance and value in diagnosis, is not dependent on our ability always to furnish a complete exposition of the mechanism of their production. Persons may differ in opinion as to the rationale of certain signs, and yet be entirely agreed respecting their special meaning and importance, the latter being based on the uniform relation found by observation to exist between the signs present during life, and the pathological changes ascertained after death. It is certainly very desirable to explain satisfactorily that connection subsisting between physical signs and physical conditions, by virtue of which the former represent the latter; but with our present knowledge, this branch of the subject of physical exploration contains many points not fully settled. ..."

The second reason was that the knowledge of acoustics and lung physiology was necessarily limited. In particular, suitable apparatus for recording acoustic events was not available which restricted the scope of any experimental work on the study of breath sounds.

Technical advances early in the 20th century helped investigators to use the acoustic science for the better understanding of lung sounds. The contributions of Martini (1921) and Martini and Müller (1923) considerably influenced the thinking of this period. Martini recorded breath sounds from the chest wall using a diaphragm and an optical capsule and determined their frequency distribution by resonance. He found the frequency band of bronchial breath sounds between 300 and 1,000 Hz. He believed that the sound was generated in the tracheo-bronchial tree in a manner similar to a wind instrument such as a flue pipe. The sound was produced by vibration of air at bifurcation points which set the air column in the adjoining

bronchus into resonance according to its natural frequency. In this way, a sound of one frequency was generated in each bronchus; low frequencies were produced in the proximal larger bronchi and progressively higher frequencies by succeeding distal bronchi, thus making up the frequency range of the bronchial breath sounds. He considered that this sound was analogous to a musical sound with a fundamental note between 300 and 550 Hz. with overtones up to 1,000 Hz. contributed by the smaller bronchi. Martini thought that the inspiratory sounds heard over the chest wall had two components; a bronchial sound of high frequency, and a vesicular sound of low frequency generated by oscillations of air containing lung tissue. During inspiration the bronchial sound was masked by the loud vesicular sound so that only the lower frequencies were audible. During expiration there was no sound in lung tissue so that only the bronchial component conducted through the lung was audible.

Bushnell (1925) however, considered that normal breath sounds were generated by vibration of the vocal cords acting as a reed. Fahr (1926) published a review of the acoustics of the bronchial breath sounds based mainly on the work of Martini and Martini and Müller. He believed that the bronchial sounds were produced in the airways like those in a wind instrument such as an organ pipe. His analysis of the tracheal breath sounds showed a frequency range between 120 and 1,000 Hz. A year earlier Cabot and Dodge (1925) found that practically all sounds of interest in auscultation were made up of frequencies below 1,000 Hz. McKusick's (1955) analysis showed frequency distribution of the vesicular breath sounds between 120 and 480 Hz. and of the tracheal breath sounds between 600 and 720 Hz.

Bullar in the late 19th century studied the conducting properties of the lung. He showed that the air containing lung was a poor conductor of sound. Martini (1921) demonstrated that the higher frequencies of breath

sounds were filtered out during transmission through the lung and the chest wall. Montgomery (1940) and McKusick (1955) arrived at similar conclusions. Buller and Dornhorst (1956) showed attenuation of higher frequencies during transmission through healthy lungs and characteristic alterations of the acoustic filtering properties of the lung in disease.

Acoustical analysis of breath sounds and attention to transmission properties of the lung and the chest wall considerably improved understanding of these sounds, but did not resolve the fundamental questions as to where and how breath sounds were generated, and the relationship of breath sounds heard at the mouth to the breath sounds heard over the chest wall. Were these sounds produced by a reed pipe system as Bushnell believed, or were they generated by a flue pipe system as proposed by Martini? If the breath sounds heard over the chest originated in the air vesicles was was commonly believed, then the mechanism of their production remained to be explained for Norris and Landis (1940) pointed out:

> "The dimensions of an alveolus is so small that there is no possibility of sound production after the manner in which it occurs in trachea or bronchi ...."

Until recently it was usual to relate abnormal lung sounds to morphological changes. Advances in pulmonary physiology and improved methods of recording acoustic events has naturally focussed attention on simultaneous functional changes in the interpretation of lung sounds. Forgacs first adopted this new approach and indicated the advantages of considering lung sounds as signals of physiological events.

The present improvement in the understanding of lung sounds is largely due to change in attitude from structure to function. Forgacs (1967) suggested the functional signifiance of adventitious lung sounds and questioned some of the more traditional assumptions on the origin of these

sounds. Nairn and Turner-Warwick (1969) showed that the intensity of breath sounds heard on the chest wall in patients with chronic bronchitis and emphysema correlated well with the regional ventilation. Leblanc, Maclem and Ross (1970) found the intensity of breath sounds recorded on the chest wall in normal subjects a good index of the rate of air entry into the underlying territory of the lung.

INTRODUCTION

Since Laennec first described breath sounds, there has been no uniform agreement on how or where they are produced. This has resulted in uncertainty on their significance in disease.

In this thesis an attempt is made to elucidate the mechanism by which breath sounds are generated. The intensity of breath sounds is related to the rate of gas flow in the airways and so to their site of production. It examines, in the light of new evidence produced, whether breath sounds on the chest wall are generated in the terminal bronchioles and the alveoli or are transmitted from the larger airways.

Laennec observed that breath sounds of dyspnoeic patients were frequently loud at the mouth. He believed that loud breath sounds in these patients originated in upper air passages and so did not reflect intrathoracic disease. This explanation was generally accepted until Forgacs (1969) suggested that loud breath sounds in patients with chronic bronchitis and asthma indicated narrowing of the larger bronchi.

The real significance of loud breath sounds at the mouth in patients with diffuse airway obstruction remained uncertain as there was no previous method available by which the breath sounds of two subjects could be compared under similar acoustic and physiological conditions. The pioneer work of Forgacs and Richardson resulted in the development of a method which allowed such comparisons to be made. The principle of the method is to record inspiratory breath sounds at a fixed distance from the mouth at instantaneous volume flow rate and to compare the sound intensity between two subjects at similar inspiratory flow rates.

Using this technique the inspiratory breath sounds of normal subjects is compared with those of patients with chronic bronchitis, asthma, primary emphysema and tracheal and bronchial stenosis. The inspiratory

sound intensity in these patients is correlated with other measurements of airways obstruction and its variation after bronchodilator and broncho-constrictor agents is studied. The clinical and the functional significance of changes in inspiratory sound intensity in these patients is indicated. Some of these observations were earlier reported by Forgacs, Nathoo and Richardson (1970). The findings published in this communication are extended and considered in greater detail in this thesis.

METHODS

1. Definition of Sound Intensity

The sound intensity is a measure of sound energy. Loudness is a subjective appreciation of the intensity of sound, but relationship between the two is not a simple one. This is because the human ear is not equally sensitive to sound intensity at all frequencies in the audible range.

Most acoustical instruments do not measure the sound intensity directly, but respond to effective sound pressure associated with a propagating sound wave. The sound intensity is proportional to square of sound pressure.

The intensity of a sound is conveniently expressed on a decibel scale and is defined by the following equation:

$$I_s = 20 \log_{10} \frac{p}{p_{ref}} \text{ dB} \qquad \text{where}$$

I_s = sound intensity

p = effective sound pressure measured

p_{ref} = reference sound pressure

dB = decibel.

It will be noted from this equation that decibel is essentially a unit implying a given ratio between the two pressures expressed on a logarithmic scale. A ratio of 2 for instance between the measured and reference sound pressures corresponds to 6 dB change, and a ratio of 4 to 12 dB change.

Because the decibel scale is based on a pressure ratio, a reference pressure must be defined. This may be any arbitrary quantity selected for convenience, or alternatively expressed in terms of an absolute pressure which is defined as the minimum sound pressure the human ear can detect and equals 0.002 dynes/cm.^{2}. For most acoustic measurements it is often sufficient to state relative changes without referring to the absolute reference intensity.

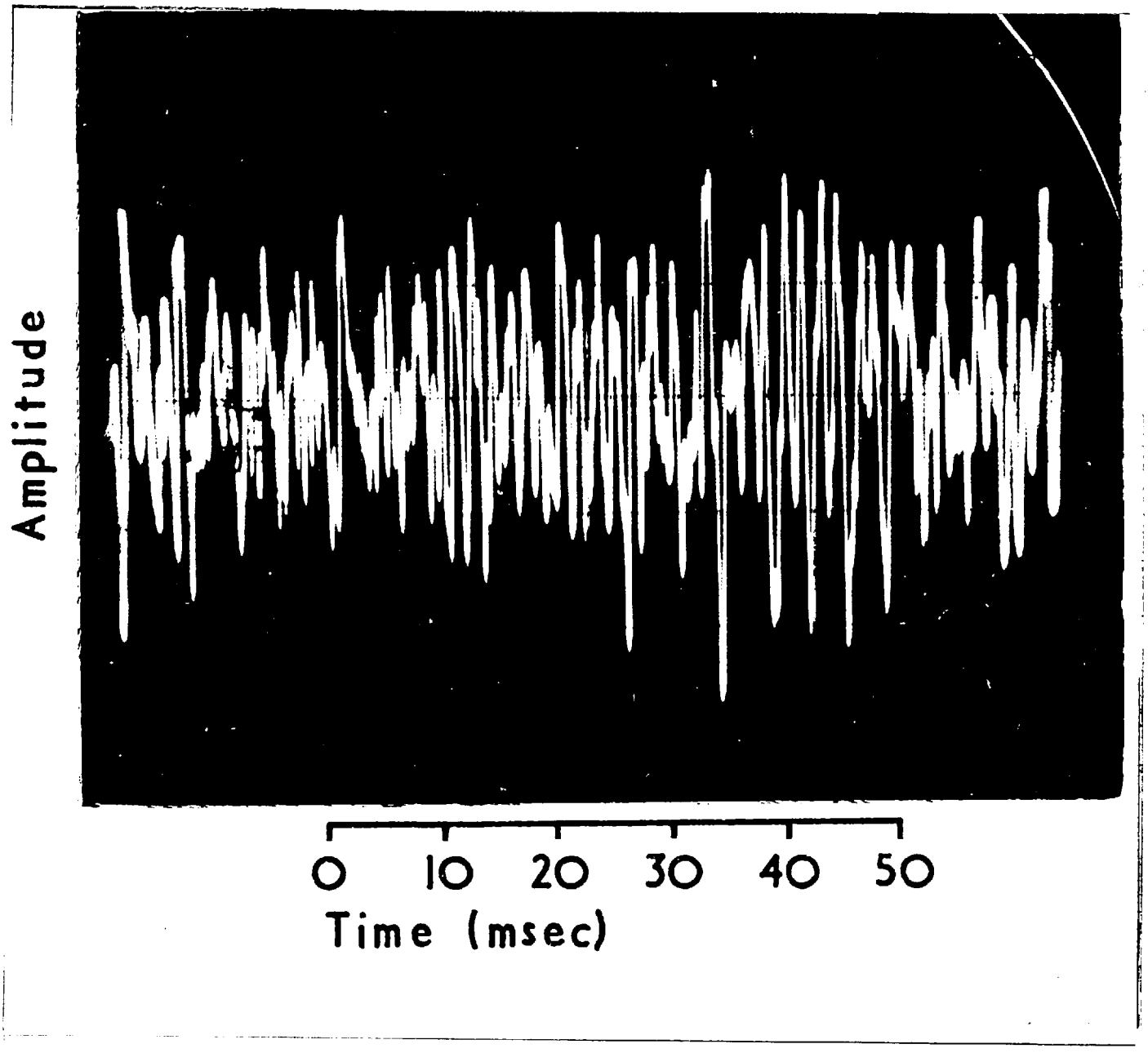


Fig 2

Inspiratory sound showing random variations in amplitude.

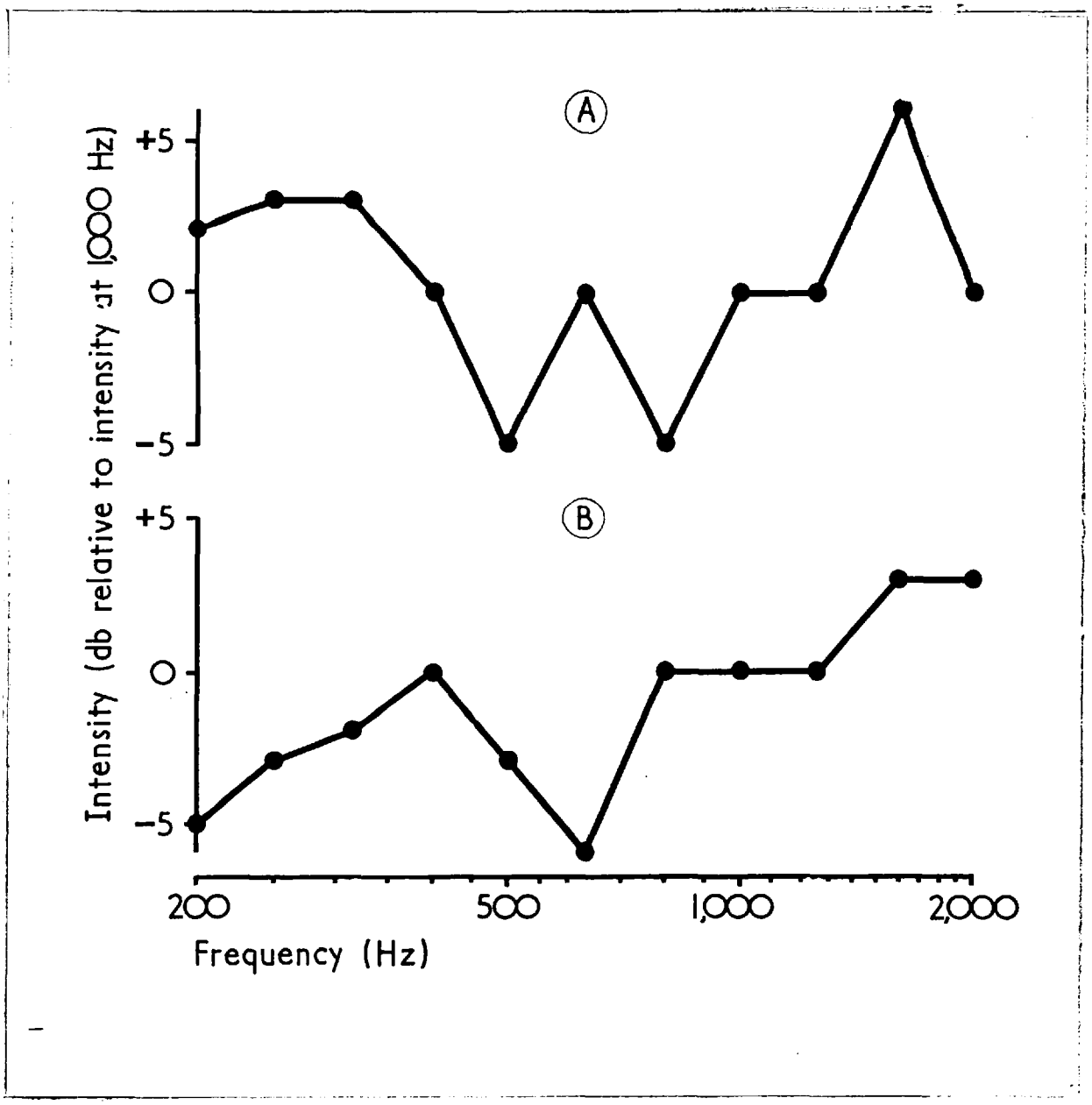


Fig 3

Spectrogram of the inspiratory sound
(A) in a normal subject and (B) in chronic bronchitis.

2. Acoustic Nature of Breath Sounds

Breath sounds are a form of noise with no definite pitch. They contain a wide range of oscillations of random amplitudes (Fig. 2). The spectrogram of breath sounds (Fig. 3) shows a fairly even and continuous distribution of sound energy between 200 and 2000 Hz. By optical analogy with white light which also shows even energy distribution as a function of frequency, the breath sounds may be referred to as a white noise.

3. Method of Recording Breath Sounds

The random nature of breath sounds makes objective comparison between two subjects under identical physiological conditions difficult. The recording method adopted in this study was developed by Forgacs and Richardson and was described in a previously published study of breath sounds (Forgacs, Nathoo and Richardson, 1971). Some of the important considerations incorporated in this method are discussed in the following sections.

(a) Location of Microphone

Breath sounds, it will be suggested (p.74) are produced in the upper air passages, trachea and the proximal intrathoracic airways. When the intensity of breath sounds at its source is of interest, there are disadvantages in recording breath sounds on the chest wall. Breath sounds recorded with a microphone placed on the chest are modified during their transmission through the lung and the chest wall. As a result the higher frequencies are filtered out and some sound energy is lost. In addition, reproducible results are difficult to obtain because small variations in applied pressure on the microphone cause large changes in sound intensity from alteration of microphone - chest wall coupling. The advantage of placing the microphone at the mouth is that breath sounds are transmitted from all the sites in a gas medium with minimum loss of sound energy. Breath sounds recorded at the mouth therefore more closely represent total sound energy generated at source.

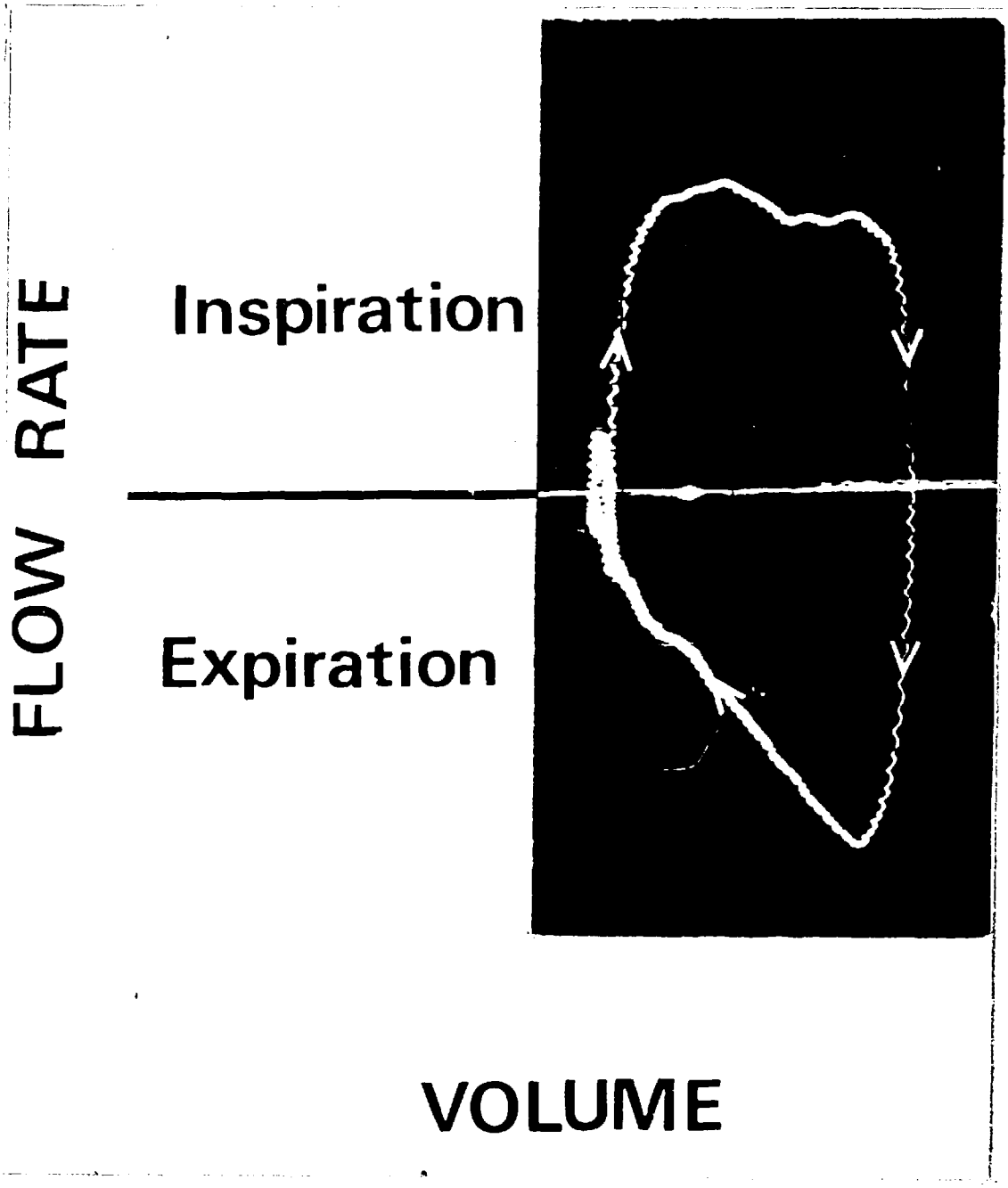


Fig 4

Inspiratory and expiratory flow-volume curves.

(b) Selection of Inspiratory Phase of Breath Sounds and Instrumental lag

Although breath sounds may be recorded in both phases of respiration, the choice was restricted to the inspiratory phase because of difficulties arising from instrumental time lag if airflow and sound intensity are to be compared.

Since the intensity of breath sounds varies with the flow rate, a comparison of breath sounds in two subjects is valid only at identical flow rates. The shape of the inspiratory flow curve shows three phases: an initial acceleration phase followed by a period of relative steady flow and a final deceleration phase. On the other hand, the expiratory flow curve has only two phases: an acceleration and a deceleration phase without an intervening period of steady flow (Fig. 4).

When breath sound intensity and airflow rate are recorded, the sound signal is displayed instantaneously while there is a significant delay before the flow rate signal is registered. The time lag between the two signals was measured in the following way. A pneumotachograph and connecting tubing were placed inside an inflated balloon and connected to a pressure transducer. The microphone was placed in close proximity of the balloon. On bursting the balloon, the sound pulse reaching the microphone triggered an oscilloscope time base and set the beam in motion. The pressure and sound signals as they appeared on the oscilloscope screen were photographed. The experiment was repeated several times and the time lag calculated was found between 40 and 50 milliseconds.

This order of time lag in the airflow signal is significant when related to the duration of acceleration or deceleration in both phases of respiration. For instance, if the time of inspiration is 2 seconds, then the duration of the acceleration and the deceleration phase is of the order of 350 milliseconds. Thus the time lag of 50 milliseconds was considered too large for breath sounds to be measured at rapidly changing flow rates.

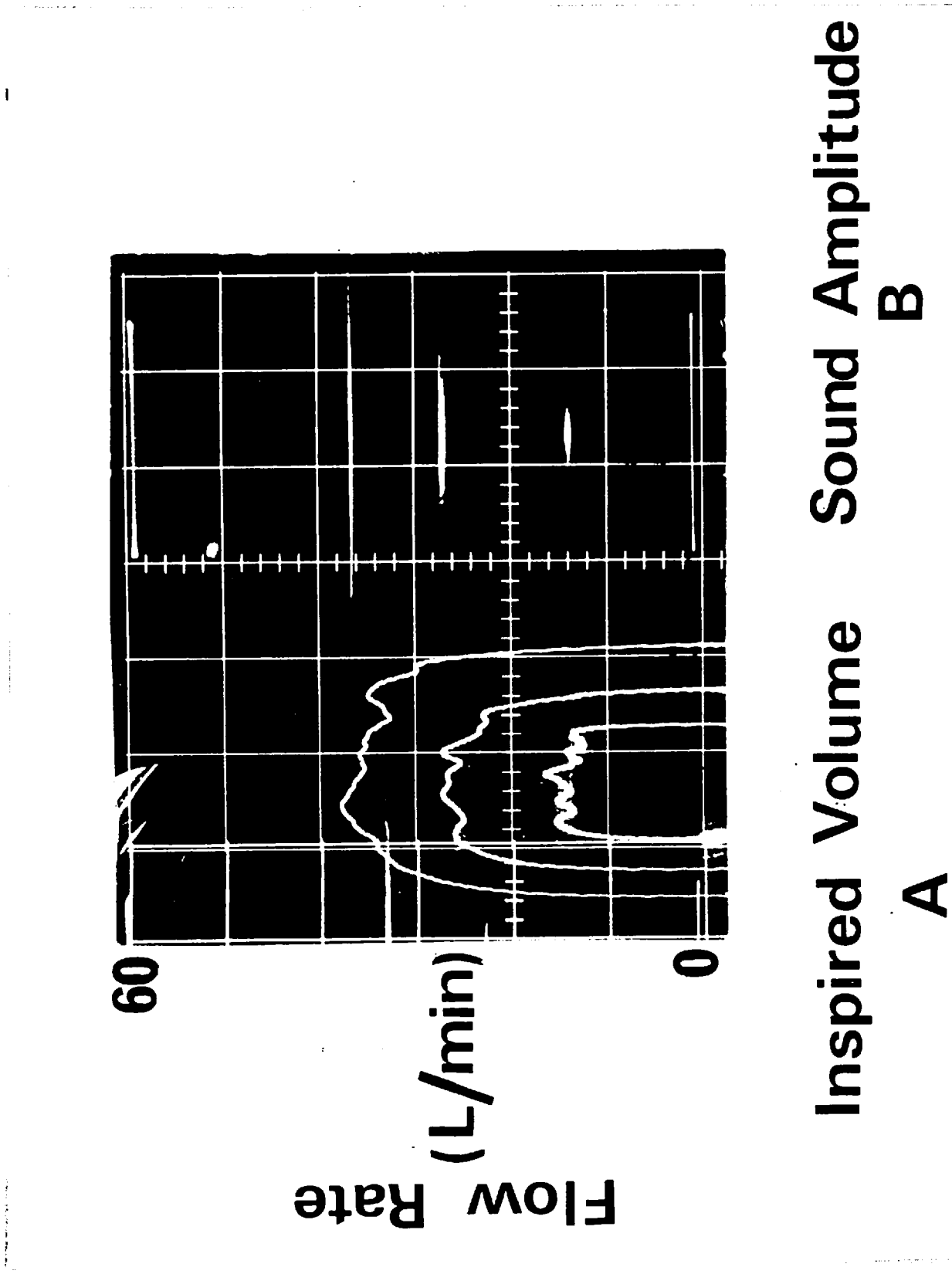


Fig 5

Sound amplitude at three different inspiratory flow rates. (A) Inspiratory flow-volume curve, (B) Inspiratory sound on X-axis, flow rate on Y-axis. Each horizontal line represents superimposed amplitudes in a sample of sound recorded during 100 msec at the corresponding plateau of the inspiratory flow rate.

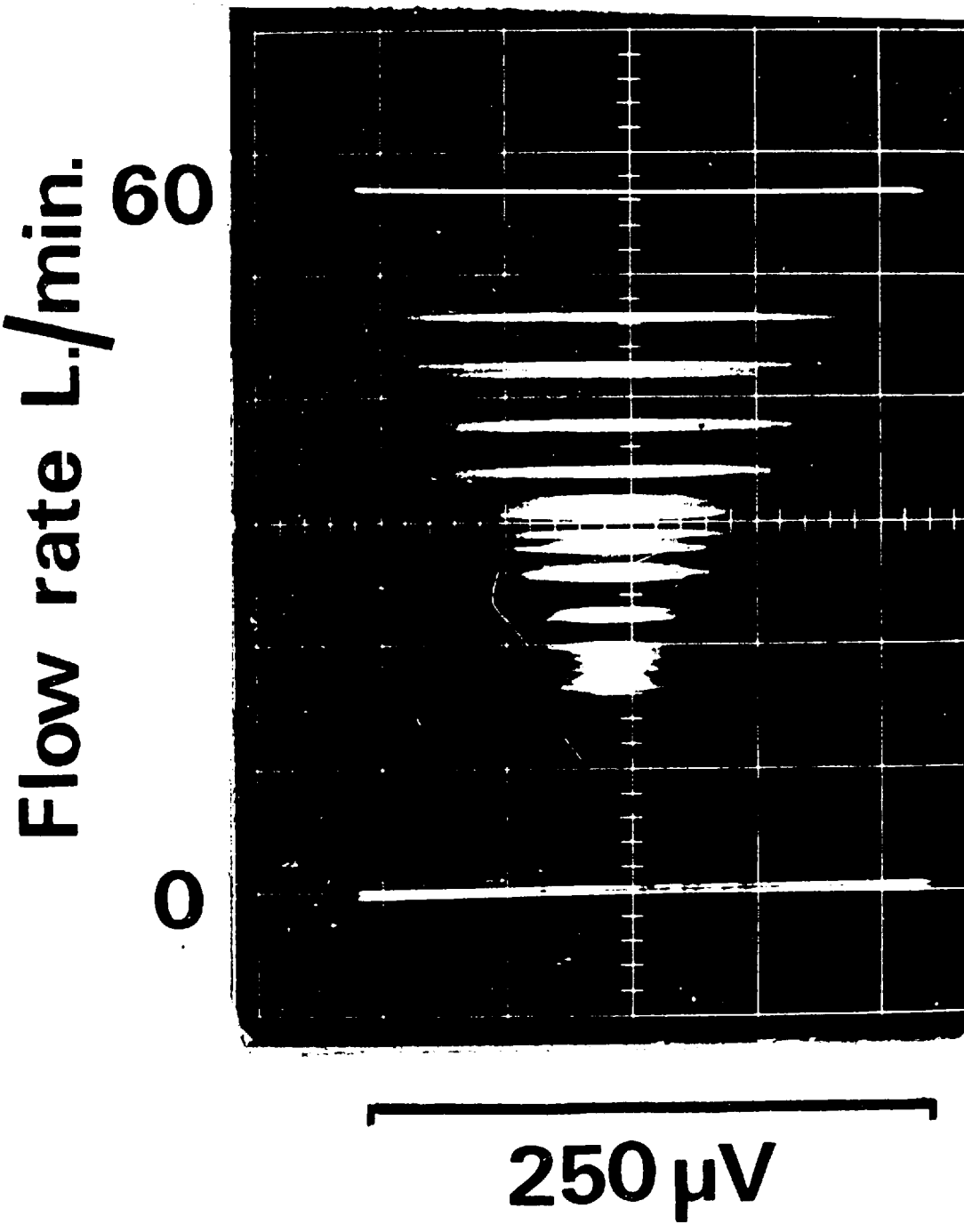


Fig 6

Composite picture of maximum amplitude
of the inspiratory sound at different flow rates.

The error due to instrumental lag however is negligible if breath sounds are recorded at a steady flow rate. The plateau of the inspiratory flow curve was the obvious choice although even here small variations of the flow occur.

(c) Display of Breath Sounds and recognition of Transient Noises

In principle breath sounds may be recorded unfiltered or as a rectified and filtered output of the microphone on a time base. The margins of random oscillations of respiratory white noise displayed on a time base are too indistinct to be suitable for measurement. The main disadvantage of recording the rectified and filtered output of breath sounds is that it masks the unwanted transient noises which may contaminate the breath sounds.

Forgacs and Richardson's method adopted in this study was to superimpose sound oscillations for a limited period at a steady flow and to display these with the flow rate on an x and y coordinates (Fig. 5). The procedure is repeated at different inspiratory flow rates and so a composite picture is obtained of sound against the corresponding flow rate (Fig. 6). During the period sound is recorded, all the oscillations large and small are superimposed and so the width of the tracing is defined by the largest amplitudes. In practice even over the plateau of the inspiratory flow - volume curve small fluctuations of the flow occur. The duration of sound recording should therefore be sufficiently long to represent average oscillations of the inspiratory white noise and the mean inspiratory flow rate at the plateau. A time of 100 milliseconds was found to be a satiefactory period to meet these two requirements.

The main advantage of this method is that as the inspiratory sound is recorded at several flow rates, a single composite picture is obtained which facilitates rapid analysis. By this method transient noises are readily detected as their margins stand out against a recognisable pattern of sound amplitudes at various flow rates.

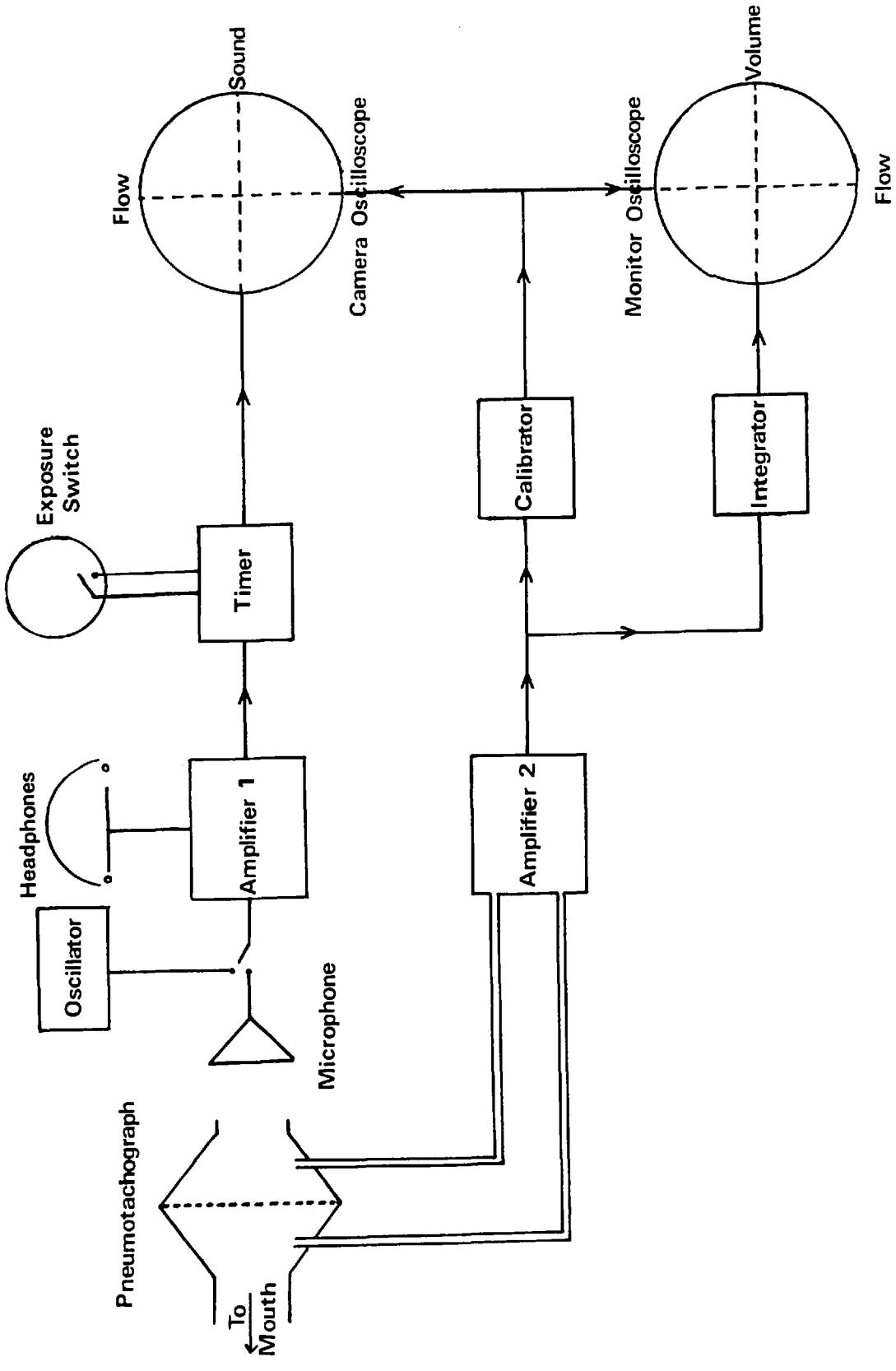


Fig 7

Instrumentation for simultaneous recording of breath sounds and flow rate at the mouth.

(d) Description of the Apparatus for measuring Inspiratory Sound Intensity

(i) General Description

This is illustrated in the block diagram (Fig. 7). A crystal microphone insert and a pneumotachograph head were mounted on the same stand 2 cm. apart. The subject was asked to breathe through a cardboard mouthpiece connected to the pneumotachograph head. The pressure difference across the mesh of the pneumotachograph was converted to electric voltages by an electromanometer. The inspiratory flow-volume curve was recorded by feeding the output of the pneumotachograph amplifier as well as its integrated output as an XY display on an oscilloscope screen.

To facilitate calibration, an electrical device was incorporated at the output end of the pneumotachograph amplifier. It was calibrated each day with the rotameter to give a defleotion of 6 cm. for a flow rate of 60 l/min. Subsequently only the electrical device was used during the day to calibrate the flow rate.

The output from the crystal microphone was amplified and displayed simultaneously with the flow rate on an oscilloscope screen fitted with a polaroid camera. A timing circuit with a hand-held push-button switch was incorporated between the amplifier and the oscilloscope. The beam of the oscilloscope screen was so arranged that it was normally off screen until the operation of the push-button switch brought the timing circuit into operation which deflected the beam onto the screen for a period of 100 milliseconds. The earphones were used to monitor the sound coming from the amplifier so that unwanted noises, such as wheezes or extraneous noises could be rejected.

(ii) Technical Details

Sound System

A crystal microphone (Acos 39 - 1 Crystal Insert) 2.5 cm. in diameter was used to record sound. The microphone was mounted on a platform

supported by a foam rubber pad to minimise vibration pickup. Attached to the same platform 2 cm. from the microphone face was the pneumotachograph head. The output of the microphone was 700 μV r.m.s. when exposed to sinusoidal sine waves of 1,000 Hz at a sound pressure of 74 dB. ** The microphone output was fed into a high gain AC preamplifier. The preamplifier incorporated a filter network to give a sharp cut off below 100 Hz. to eliminate unwanted hum and low frequency ambient noise. The frequency response of the entire sound system including the microphone was within 3 dB between 300 to 2,000 Hz. The preamplifier was set so that an input of 250 μV (r.m.s.) 1,000 Hz. sine wave from the signal generator gave a deflection of 5 cm. on the oscilloscope screen. A polaroid camera (Telfoid Polaroid Camera) was used to photograph sound.

Flow Rate System

The flow rate was measured while the patient breathed through a cardboard cylinder 9.2 cm in length and 2.8 cm in diameter attached to the pneumotachograph head (Mercury Electronics Flow Head Type F.1). The pressure changes across the mesh of the pneumotachograph were translated into electrical voltages by a defocussing electromanometer (Mercury Electronics Type M3 - A10 Capsule) with an output of 650 mV for a flow of 50 l/min. The output of the electromanometer was fed into an integrating device to obtain simultaneous flow volume curve which was displayed on the monitor oscilloscope screen (Telequipment S52). At the same time the flow rate and sound amplitude were displayed as an XY plot on the main oscilloscope (Telequipment D43R). The pneumotachograph head had a linear output flow response up to 300 l/min.

** mV = millivolt i.e. 10^{-3}volt

μV = microvolt i.e. 10^{-6}volt

r.m.s = root mean square value of the amplitude of a sine wave.

An additional electrical device was incorporated at the output end of the electromanometer to facilitate flow calibration following initial setting up with a rotameter. The sensitivity was arranged so that 60 l/min. flow rate gave a 6 cm. deflection on the oscilloscope.

(e) Procedure of Sound Recording

The apparatus was first calibrated to obtain appropriate sound and the flow rate deflections. With the noseclip on the patient was asked to breathe naturally through a cardboard cylinder attached to the pneumotachograph head. While observing the monitor screen the operator first noted the resting inspiratory flow rate. Opening the shutter of the camera and still observing the monitor screen the operator pressed the push-button switch during steady flow. This deflected the beam on the screen on the main oscilloscope for a period of 100 milliseconds, thus exposing the film picture of superimposed sound amplitudes corresponding to the flow rate. By instructing the subject to take varying size breaths, and with the shutter still open, a composite picture was built up by repeating the procedure at various flow rates up to 60 l/min. (Figs. 4 and 5).

In the event of unwanted noises being heard through the earphones the operator did not make exposures until these sounds subsided. The procedure was completed by recording both the standard sound and the flow rate signals. The photograph was then removed from the camera for analysis. The whole procedure was carried out in a quiet, but not soundproof room and usually took only a few minutes. When the drug effect was studied the procedure was repeated in exactly the same way after appropriate time interval.

Breath Sounds on the Chest Wall

Breath sounds on the chest wall were recorded with a microphone fitted in an aluminium cup with a circular suction chamber (S.E. Lab., Type 4/84) fixed on the posterior chest wall 2 cm above the diaphragm. Simultaneous flow rate was measured with a pneumotachograph connected to an electromanometer (Mercury Electronics Type M3 - A10 Capsule). The amplified output of the

microphone and the pneumotachograph amplifier were displayed on two channels of a UV recorder (S.E. UV Galvanometer multichannel recorder). The frequency response of the microphone, amplifier and the recording system was linear between 100 and 2,000 Hz.

The sequence of gas as breathed in the helium experiment was air, oxygen and a mixture of 79% He and 21% O_2. During this period the microphone remained fixed on the chest wall.

The breath sounds of patients with cogwheel breathing were first recorded on a tape recorder (Ferrograph Series Seven, Mk II) and subsequently displayed on a UV recorder. The microphone was placed near the apex of the heart.

Maximum Mid-inspiratory Flow Rate (MMIF)

This measurement was usually done after recording the inspiratory breath sounds. The inspiratory flow-volume curve was displayed on the monitor screen as previously described. Breathing through a cardboard cylinder and with the noseclip on the subject was asked to make a forced inspiratory effort from the position of full expiration. The flow rate corresponding to mid-inspiratory volume was noted. The highest of three efforts was recorded.

Airway Resistance (R_{aw})

The inspiratory airway resistance was measured in a constant pressure body plethysmosgraph by the method of Dubois, Botelho and Comroe, 1956. The airway resistance slope was measured by displaying pressure changes in the box and flow rate as an XY plot, and intrathoracic gas volume slope by displaying mouth and box pressure changes as an XY plot on an oscilloscope screen. At least three satisfactory recordings were obtained with the subjects panting at not more than 0.5 l/sec. From the airway resistance and the intrathoracic gas volume slopes respectively were calculated inspiratory airway resistance (R_{aw}), intrathoracic gas volume (Vtg)

and the specific airway resistance (SR_{aw} = Raw x Vtg). The average of three determinations was used, and the variations of slope in three recordings were not more than 2 degrees.

Peak Expiratory Flow Rate (PEF)

A Wright peak flow meter was used to measure peak expiratory flow rate. The highest of the three determinations was recorded.

Forced Expiratory Volume in one second (FEV_1) and Vital Capacity (VC)

These were recorded on a Vitalograph spirometer calibrated against a standard water spirometer. All records were obtained from a position of full inspiration. The highest of the three recordings corrected to BTPS was used.

Selection of Patients

Normal Subjects

The normal subjects selected were six adult men and six adult women (ages 21 to 40, mean 30 years). None of these subjects had a past history of any serious chest illness or complained of chronic cough, expectoration or breathlessness. All had FEV_1 and VC in excess of 80% of predicted values and an FEV_1/FVC ratio greater than 75%.

Chronic Bronchitis

Fifty six men and two women (ages 44 to 77, mean 61.6 years) were studied. All these patients had a history of chronic cough and expectoration and evidence of diffuse airway obstruction (mean FEV_1 53% of predicted values, mean FEV_1 52% of FVC).

Asthma

Fifty subjects were studied, twenty seven men and twenty three women (ages 16 to 68 years), mean 42.6 years). All patients showed reversibility of airways obstruction either spontaneously or under treatment. This group of patients included those with early and late onset of asthma.

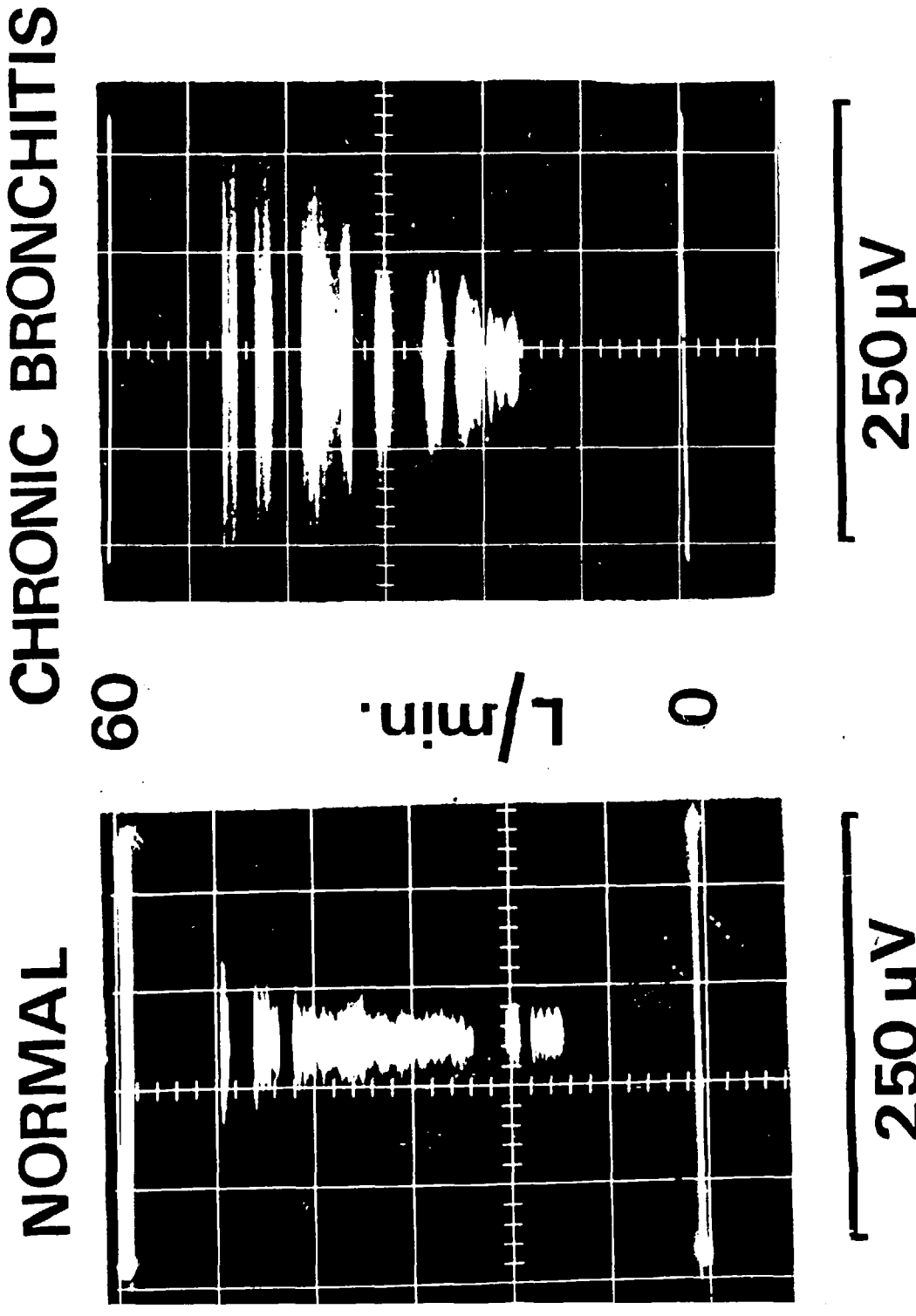


Fig 8

Comparison of the rate of increase of inspiratory sound Amplitude in relation to flow rate in a normal subject and in chronic bronchitis.

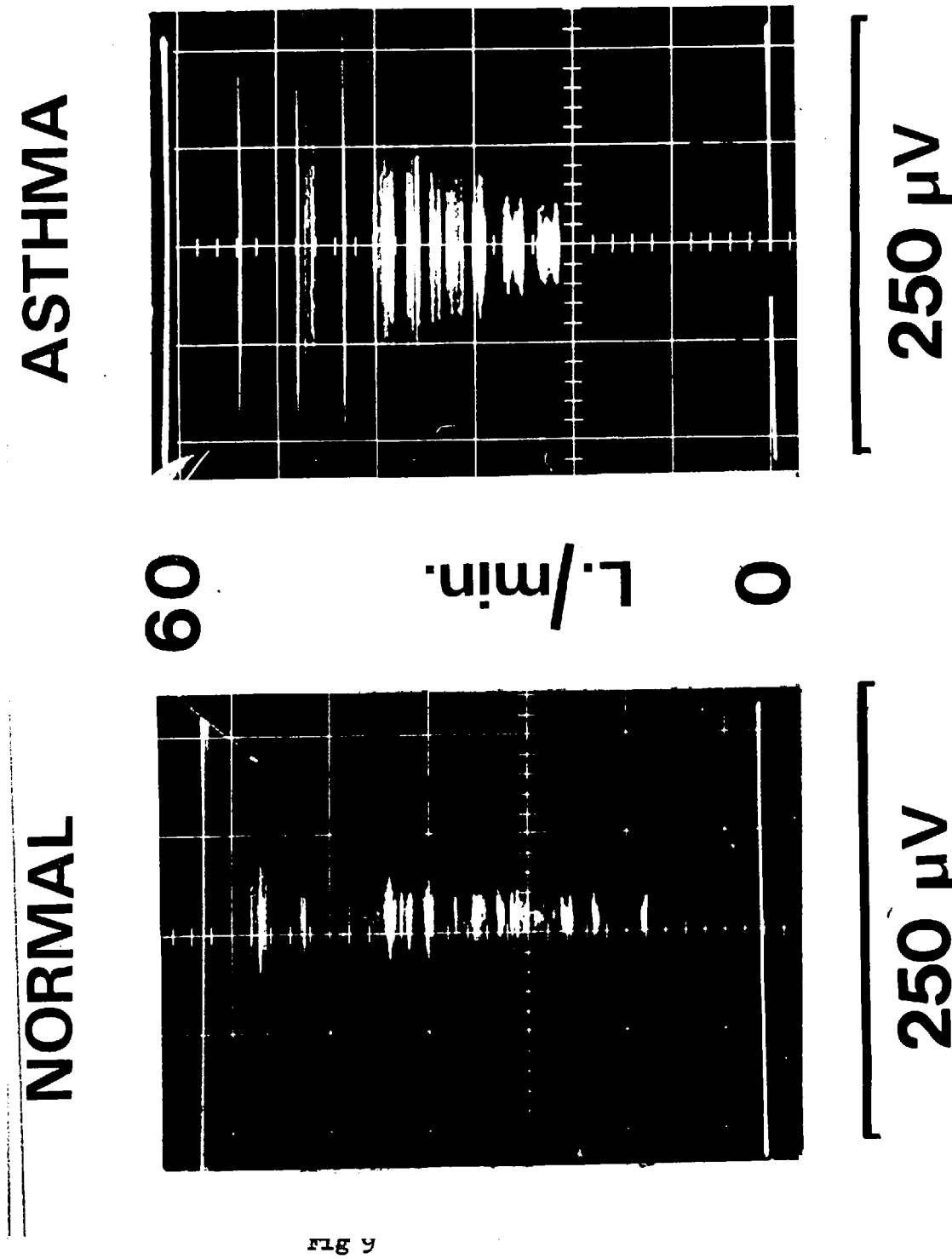


Fig 9

Comparison of the rate of increase of inspiratory sound amplitude in relation to flow rate in a normal subject and in asthma.

In a few of these asthmatic patients airways obstruction was irreversible at the time of study although they showed reversibility at some time during the course of their illness.

Primary Emphysema

Six men and one woman (ages 40 to 65, mean 54.6 years) were studied. All seven patients had a characteristic radiological appearance and functional abnormalities of emphysema without a previous history of cough or sputum. Two of these patients had α_1 - antitrypsin deficiency.

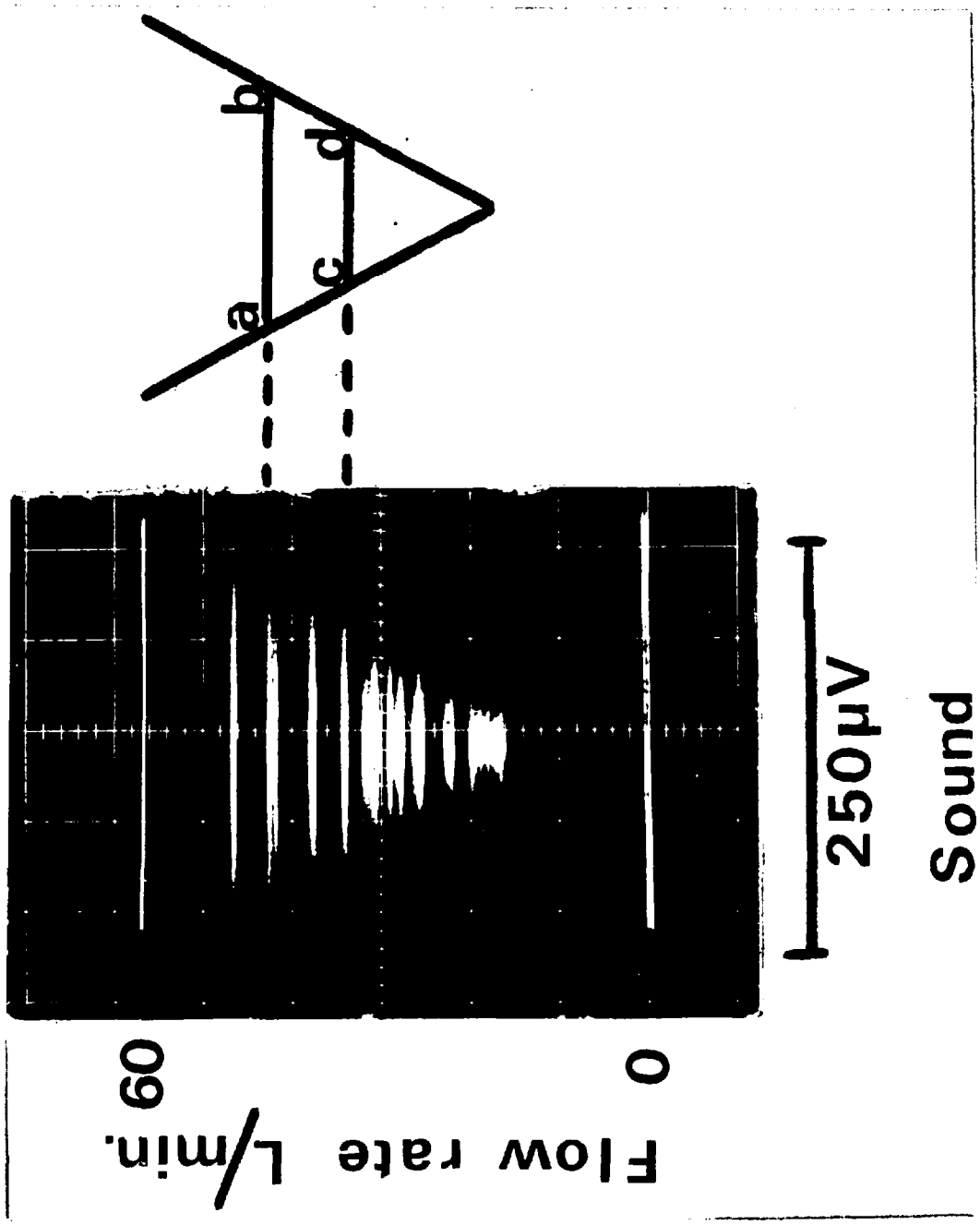
OBSERVATIONS

Comparison of Normal and Abnormal Breath Sounds

At inspiratory flow rates between 10 - 20 l/min. the breath sounds of healthy subjects were too faint to appear above the ambient noise. At higher flow rates up to about 60 l/min. the correlation between the inspiratory sound and the flow rate was approximately linear. Observations on 12 normal subjects, each tested on six different occasions showed that the inspiratory sound varied little between the subjects or from one day to another in the same subject (Tables I and II).

At an inspiratory flow rate of 60 l/min. the mean output of the microphone was 52.4 μv (Table I). These observations defined the normal inspiratory sound at the mouth in acoustic terms as a white noise of 51 dB intensity at a flow rate of 60 l/min. calculated from the measured output of the microphone of 700 μv r.m.s. when exposed to sinusoidal sine wave of 1000 Hz at a sound pressure of 74 dB.

In chronic bronchitis and asthma, as in normal subjects, the inspiratory breath sounds emerge from the ambient noise at a flow rate between 10 and 20 l/min. Above this flow rate the correlation with sound intensity was again approximately linear, but compared with normal subjects the increase in the output of microphone for corresponding increments in the flow rate was usually much greater (Figs. 8 and 9).



Measurement of the inspiratory sound intensity (I_S). The rate of expansion of inspiratory sound amplitude per 10 l/min is ab - cd = 55 μV

$$I_S = 20 \log \frac{55}{10} = 15 \text{ dB}$$

Although in principle abnormally loud breath sounds may be compared with the normal at any single flow rate, in practice the choice was limited to an inspiratory flow rate of 60 l/min. because the microphone output at lower flow rates in normal subjects was usually too small to be readily measured. On the other hand, the patients with asthma and chronic bronchitis were often unable to achieve the inspiratory flow rate of 60 l/min. during quiet breathing. For this reason it was found more convenient to compare the rate of increase of sound above the ambient noise between the two groups.

Table II shows the rate of increase in sound expressed in electrical terms as a rise in microphone output per 10 l/min. increment in inspiratory flow rate in 12 normal subjects each tested on six different occasions. The mean output increment of 10 uv per 10 l/min. was defined as the reference point against which all breath sounds were compared. The intensity of abnormally loud breath sounds was calculated by comparing the rate of increase in the output of microphone with the normal standard, as defined above, and expressing their ratio on a logarithmic scale in decibels above normal. Details of the calculation are as follows (Fig. 10):

p_1 = rate of increase of the output of the microphone per 10 l/min. flow rate increment in normal subjects (i.e. 10 μV per 10 l/min).

p_2 = rate of increase of the output of the microphone per 10 l/min. flow rate increment of breath sounds under comparison.

I_s = intensity of breath sounds under comparison.

$$I_s = 20 \log \frac{p_2}{p_1}$$

Example: The intensity of breath sounds which increase the output of the microphone by 40 μV per 10 l/min flow is $20 \log \frac{40}{10} = 12$ dB above normal.

An assumption made in this calculation is that the linear increase in the intensity of all normal and abnormal breath sounds starts from the same flow rate. In fact the flow rates at which the breath sounds emerge from the ambient noise vary between 10 and 20 l/min. A single figure indicating the difference in intensity between normal and abnormal inspiratory sounds may therefore be accurate only at one particular flow rate. It is however a reasonable and convenient approximation for all flow rates within the physiological range.

Subjective error in the measurement of sound intensity may arise if the margins of the sound amplitudes at various flow rates are not well defined. In this instance, the observer may draw the triangle of the intensity flow rate relationship too wide or too narrow and so overestimate or underestimate the inspiratory sound intensity. The magnitude of this error was assessed by asking three observers to calculate the sound intensity from fifty recordings independently. The results are shown in Table III. The mean difference in sound intensity between two observers was less than a decibel. The subjective error, however, for the same observer was constant. For example, observer A consistently overestimated sound intensity compared to observer C. These differences in sound intensity due to measurement error was considered to be small.

Chronic Bronchitis and Asthma

The mean inspiratory sound intensity in fifty six patients with chronic bronchitis was 15 dB (range 0-28 dB, S.D. 7.0) and in fifty asthmatic subjects was 13 dB (range 0-25 dB, S.D. 7.0).

The inspiratory sound intensity in patients with chronic bronchitis and asthma was correlated with the peak expiratory flow rate (PEF), forced expiratory volume in one second (FEV_1), vital capacity (VC), airway resistance (R_{aw}), and the maximum mid-inspiratory flow rate (MMIF). The results are shown on Table IV.

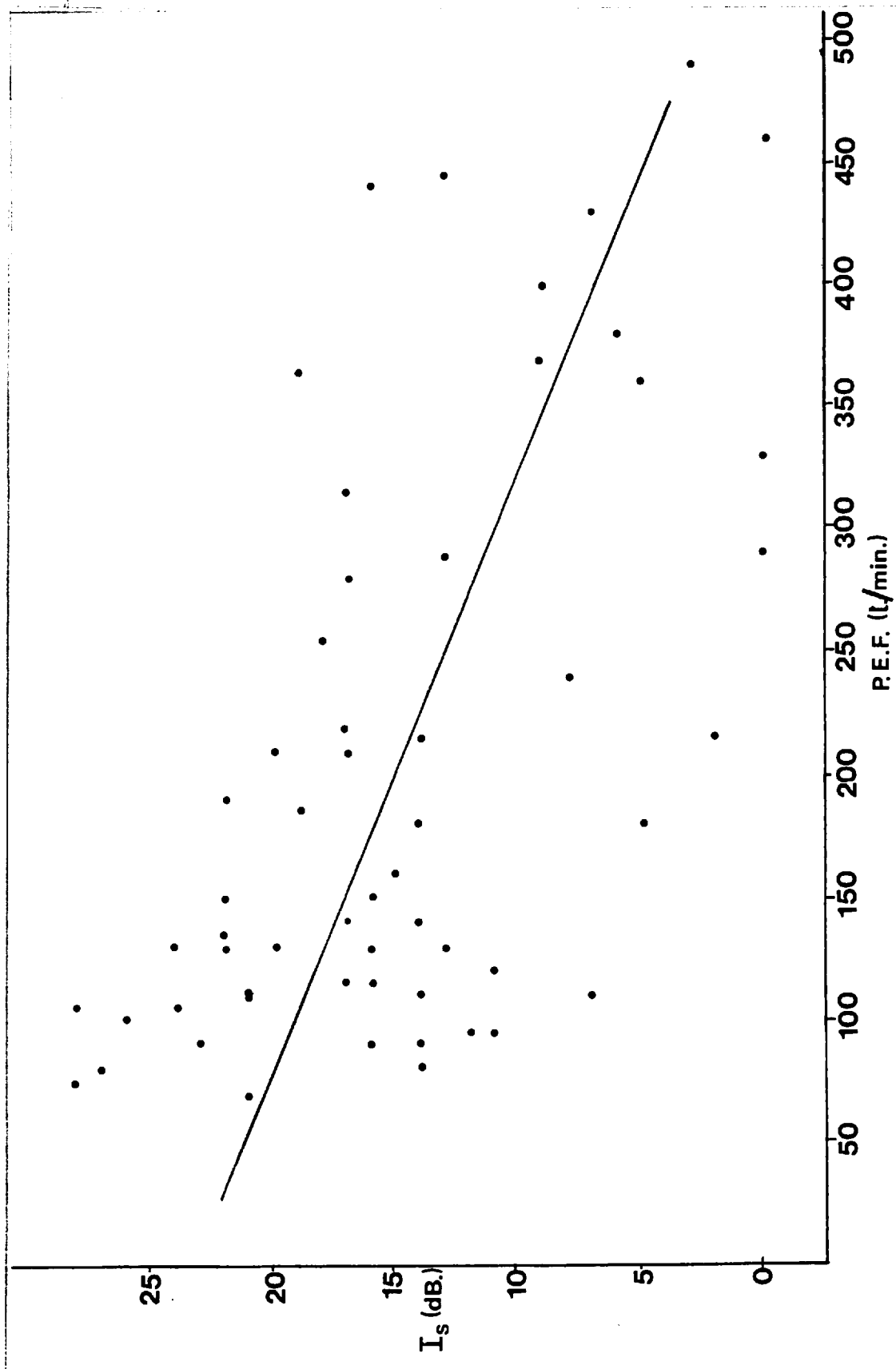


Fig 11

Scatter diagram of correlation between the inspiratory sound intensity and the peak expiratory flow rate in chronic bronchitis. ($r = -0.60$, $P < 0.001$).

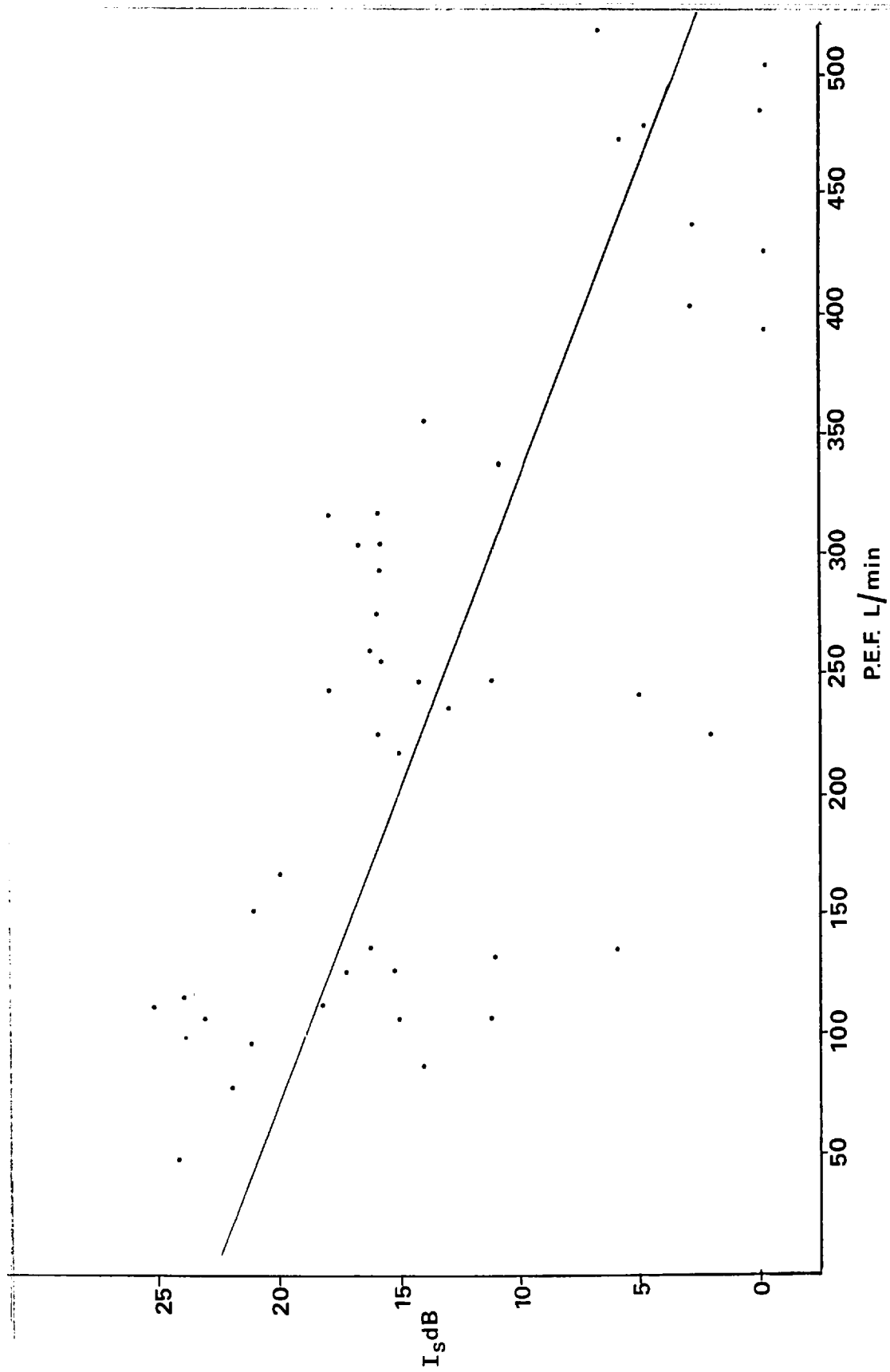


Fig 12

Scatter diagram of correlation between the inspiratory Sound intensity and the peak expiratory flow rate in asthma ($r = -0.56$, $P < 0.001$).

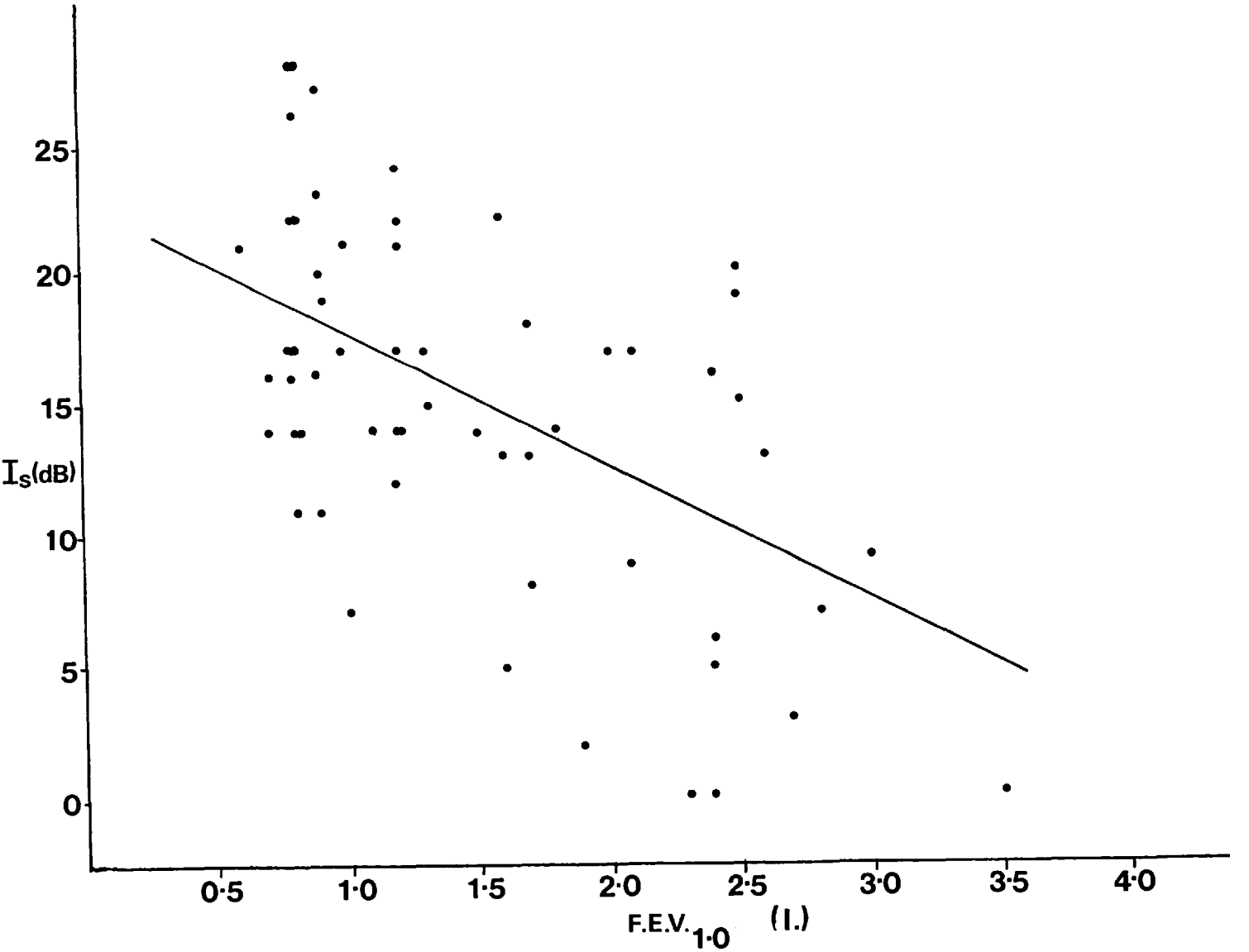


Fig 13

Scatter diagram of correlation between the inspiratory sound intensity and the FEV_1 in chronic bronchitis ($r = -0.60$, $P < 0.001$).

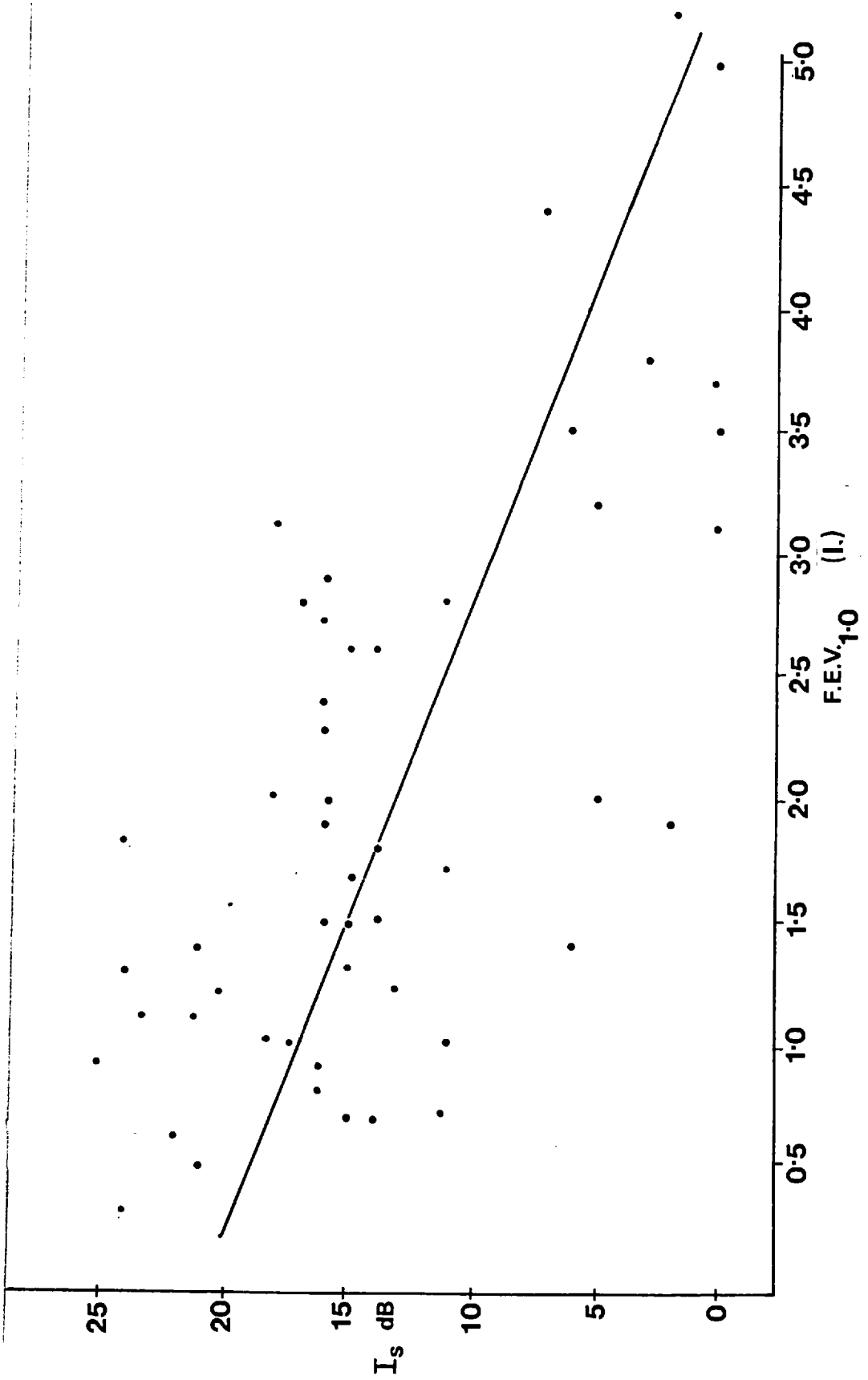


Fig 14

Scatter diagram of correlation between the inspiratory sound intensity and the FEV_1 in asthma ($r = -0.63$, $P < 0.001$).

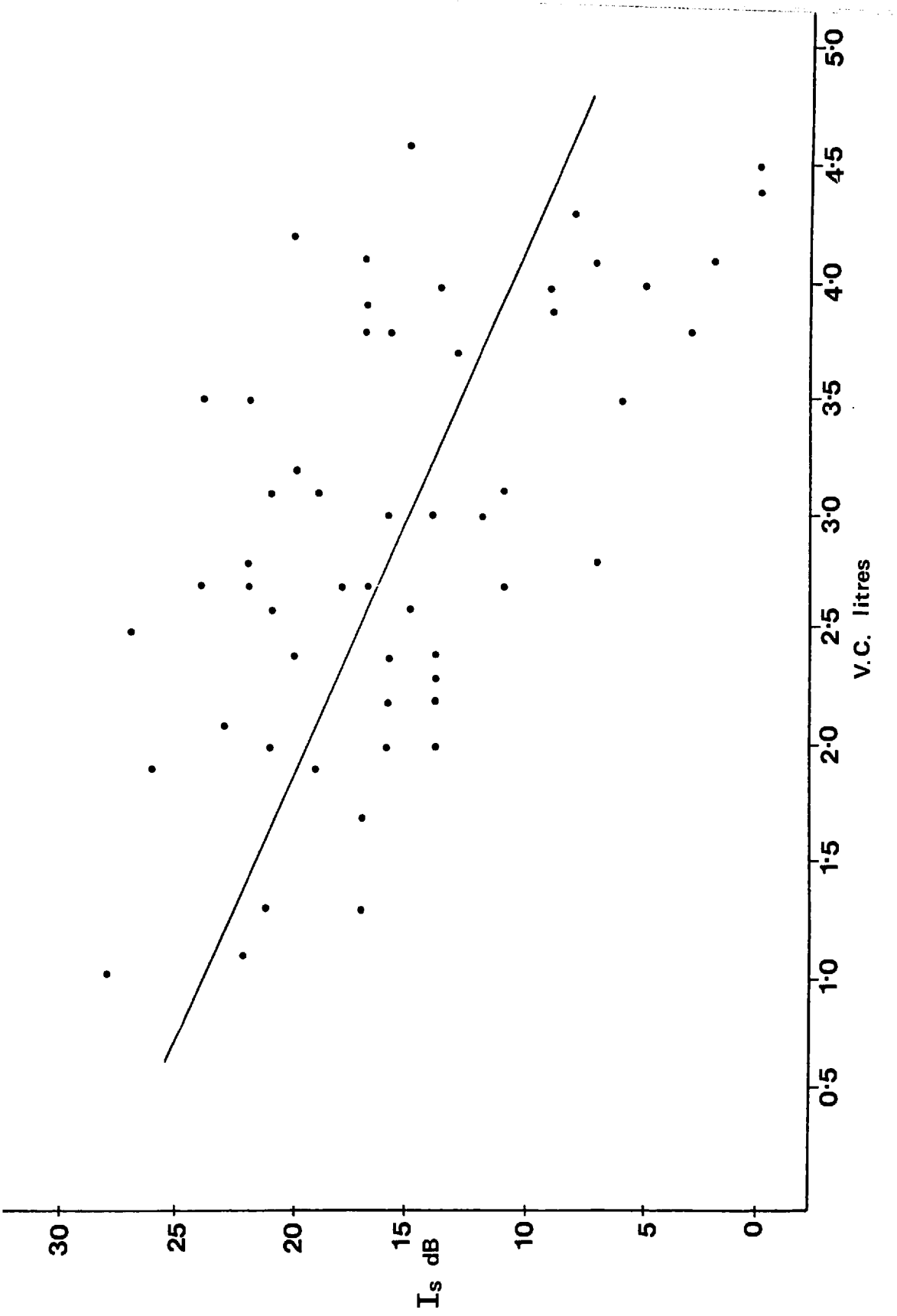


Fig 15

Scatter diagram of correlation between the inspiratory sound intensity and the vital capacity (r = -0.63, $P < 0.001$) in chronic bronchitis.

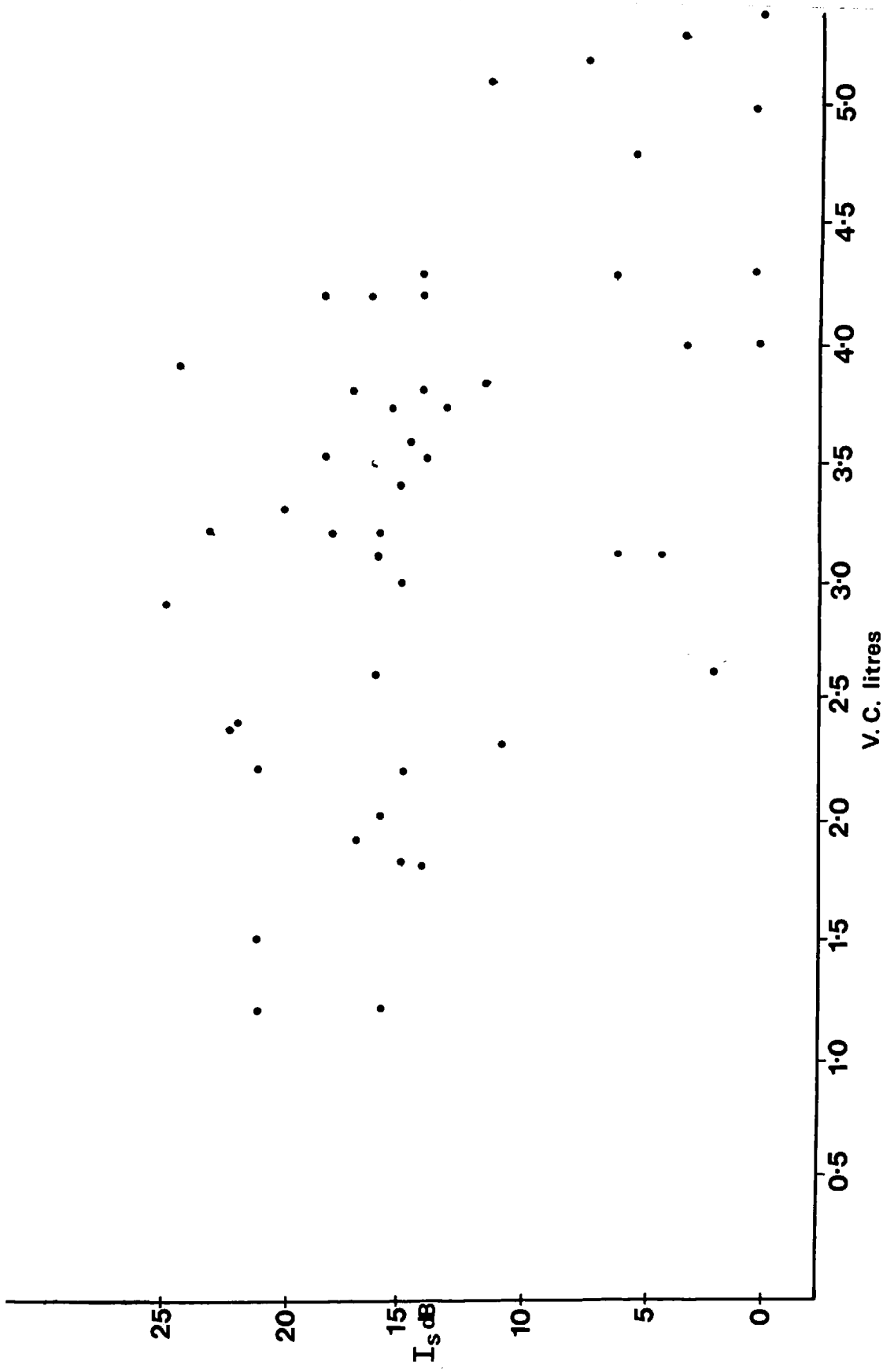


Fig 16

Scatter diagram of correlation between the inspiratory sound intensity and the vital capacity in asthma ($r = -0.44$, $P<0.01$). Poor correlation due to disproportionately loud breath sounds for the corresponding VC in some patients.

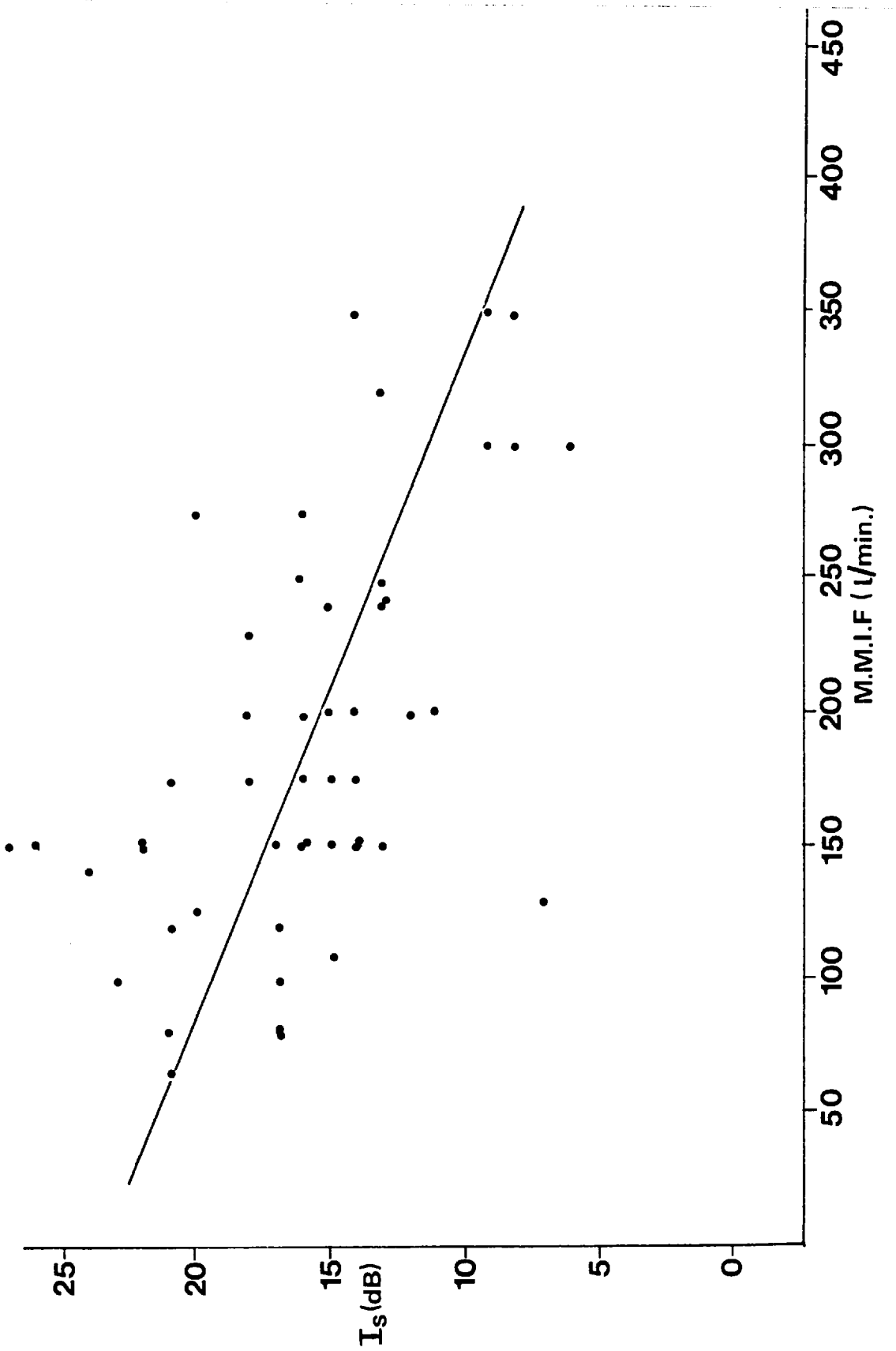


Fig 17

Scatter diagram of correlation between the inspiratory sound intensity and the maximum mid-inspiratory flow rate in chronic bronchitis. ($r = -0.61$, $P < 0.001$).

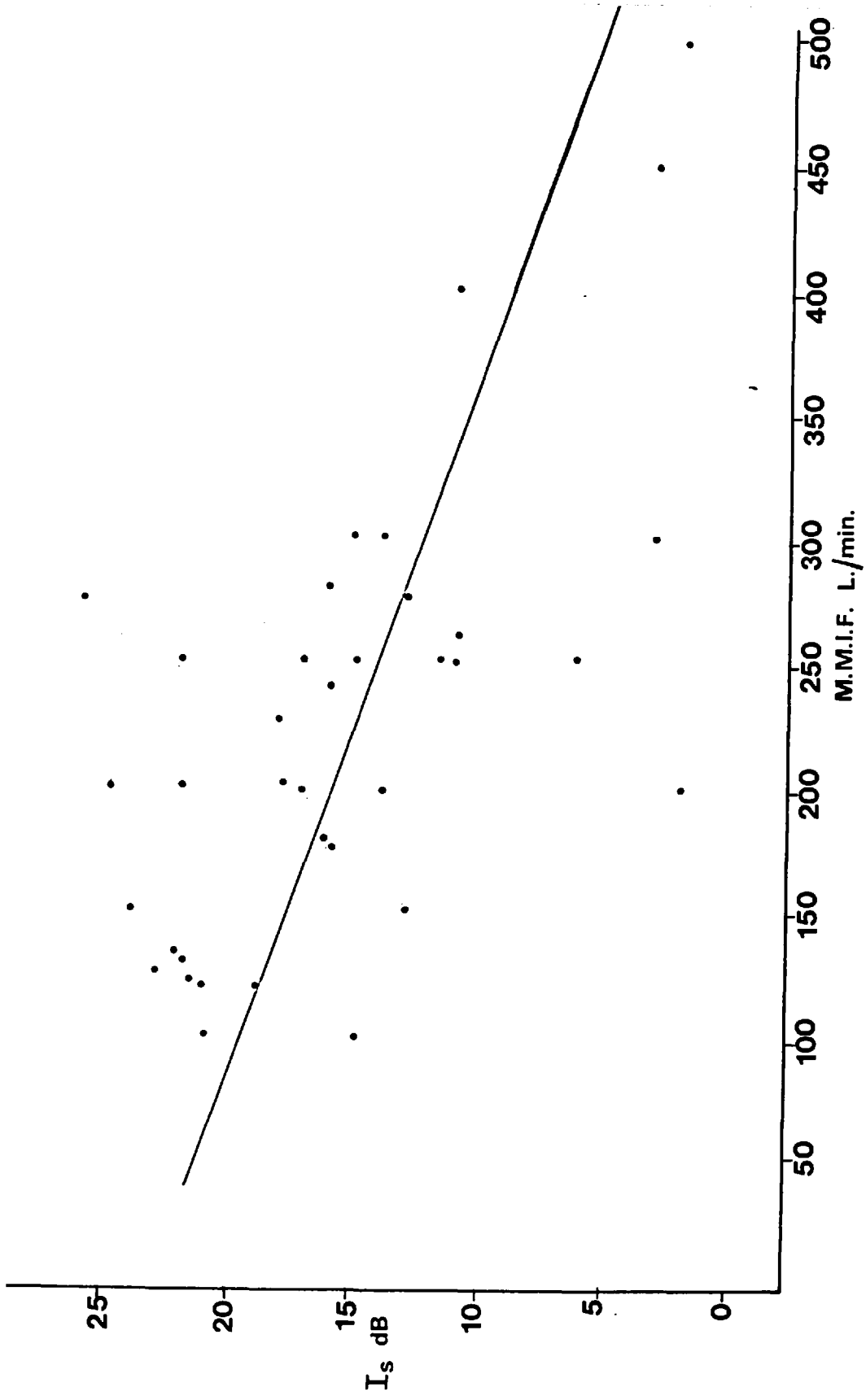


Fig 18

Scatter diagram of correlation between the inspiratory sound intensity and the maximum mid-inspiratory flow rate in asthma. (r = - 0.53, $P<0.01$).

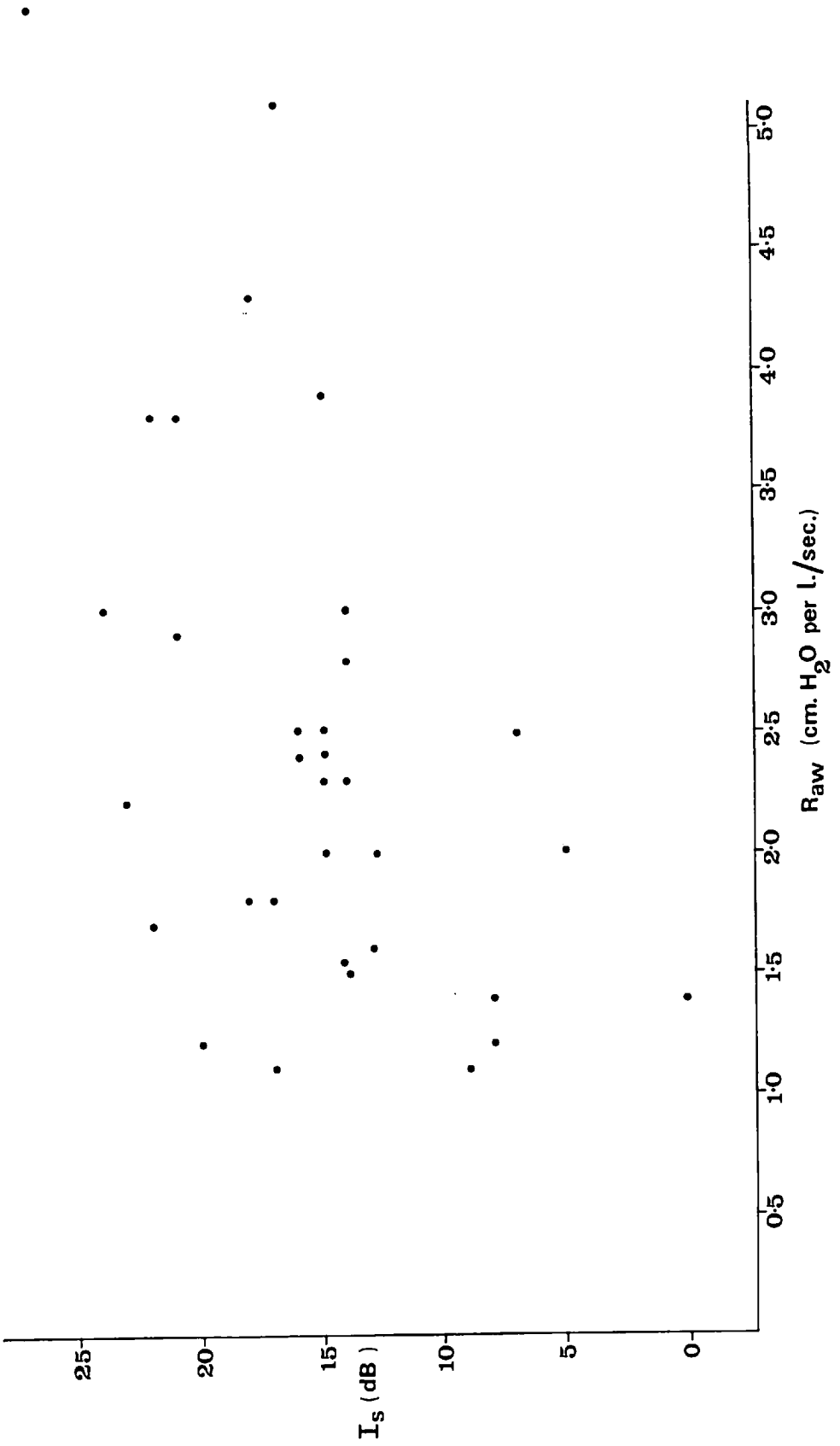


Fig 19

Scatter diagram of correlation between the inspiratory sound intensity and the airway resistance in chronic bronchitis. ($r = + 0.50$, $P < 0.01$).

In patients with chronic bronchitis and asthma, a good inverse correlation was found between the inspiratory sound intensity, PEF (Figs. 11 and 12) and the FEV_1 (Figs. 13 and 14).

The correlation between the inspiratory sound intensity and the vital capacity was good in patients with chronic bronchitis (Fig. 15) but poor in asthmatic subjects (Fig. 16). The significance of this difference is discussed later (P. 77).

The inspiratory sound intensity correlated well with the maximum mid-inspiratory flow rate in chronic bronchitis and asthma (Figs. 17 and 18).

The correlation between inspiratory sound intensity and the inspiratory airway resistance in chronic bronchitis although statistically significant was poor compared to PEF, FEV_1 and the MMIF (Fig. 19). In asthma the correlation between the two measurements was not significant. The correlation was not further improved when the inspiratory sound intensity was compared with the specific airway resistance or with the logarithm of airway resistance and the logarithm of specific airway resistance (Table V).

Primary Emphysema

The inverse correlation between the intensity of the inspiratory breath sounds and the PEF and FEV_1 found in bronchitis and asthma was not seen in primary emphysema. All seven patients had either normal or slight increase in inspiratory sound intensity despite severe expiratory obstruction shown by the low PEF and FEV_1 (Figs. 20, 21 and Table VI). The significance of this observation in relation to site of airways obstruction is considered later (p. 76).

Bronchial and Tracheal Stenosis

In five patients with bronchial stenosis and one patient with tracheal stenosis, the inspiratory breath sounds were louder than predicted from the forced expiratory tests (Fig. 21, Table VII). The inspiratory sound intensity of a patient with cicatricial tracheal stenosis was measured

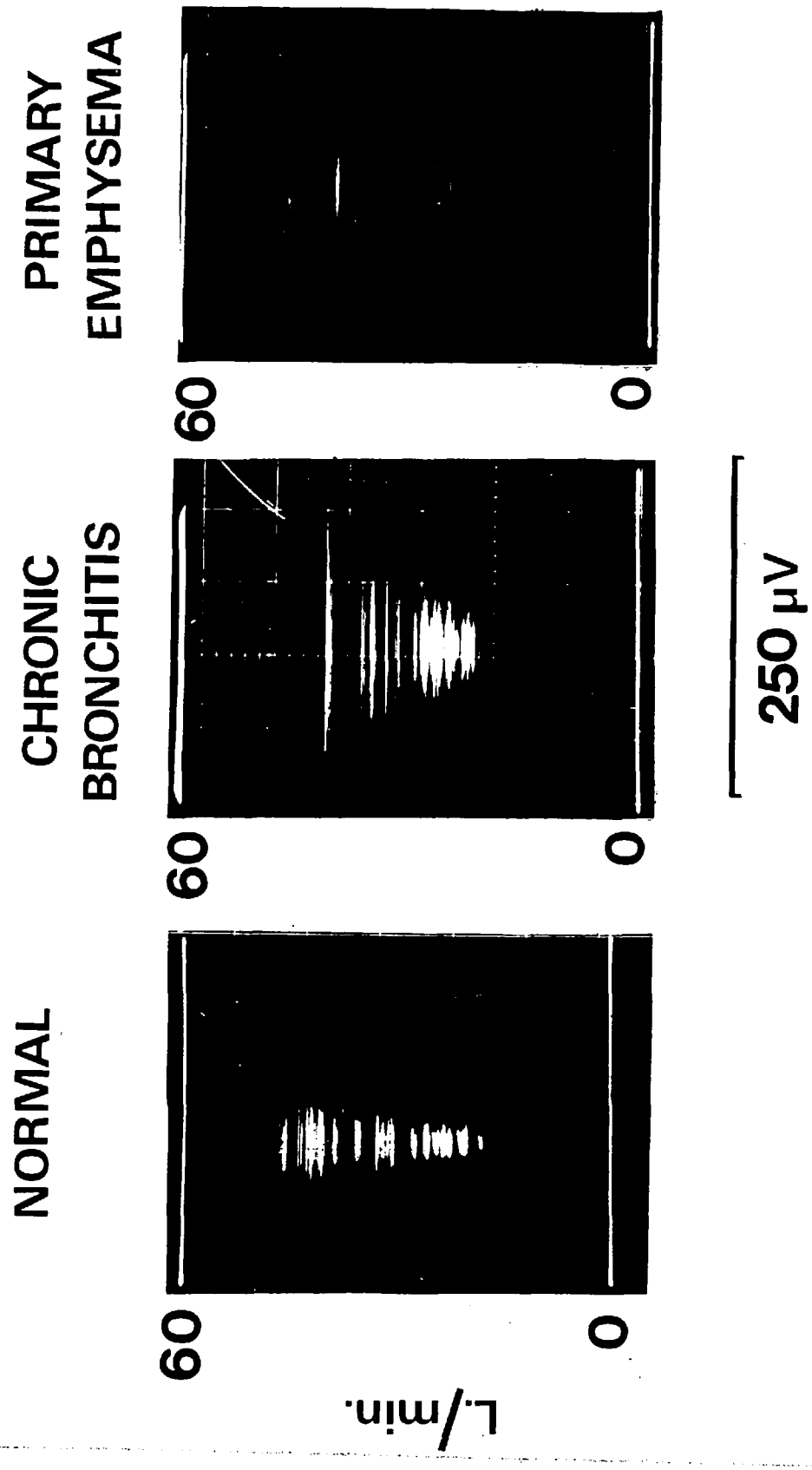


Fig 20

Inspiratory breath sounds in health (FEV_1 3.9L), chronic bronchitis (FEV_1 1.1L) and primary emphysema (FEV_1 0.8L)

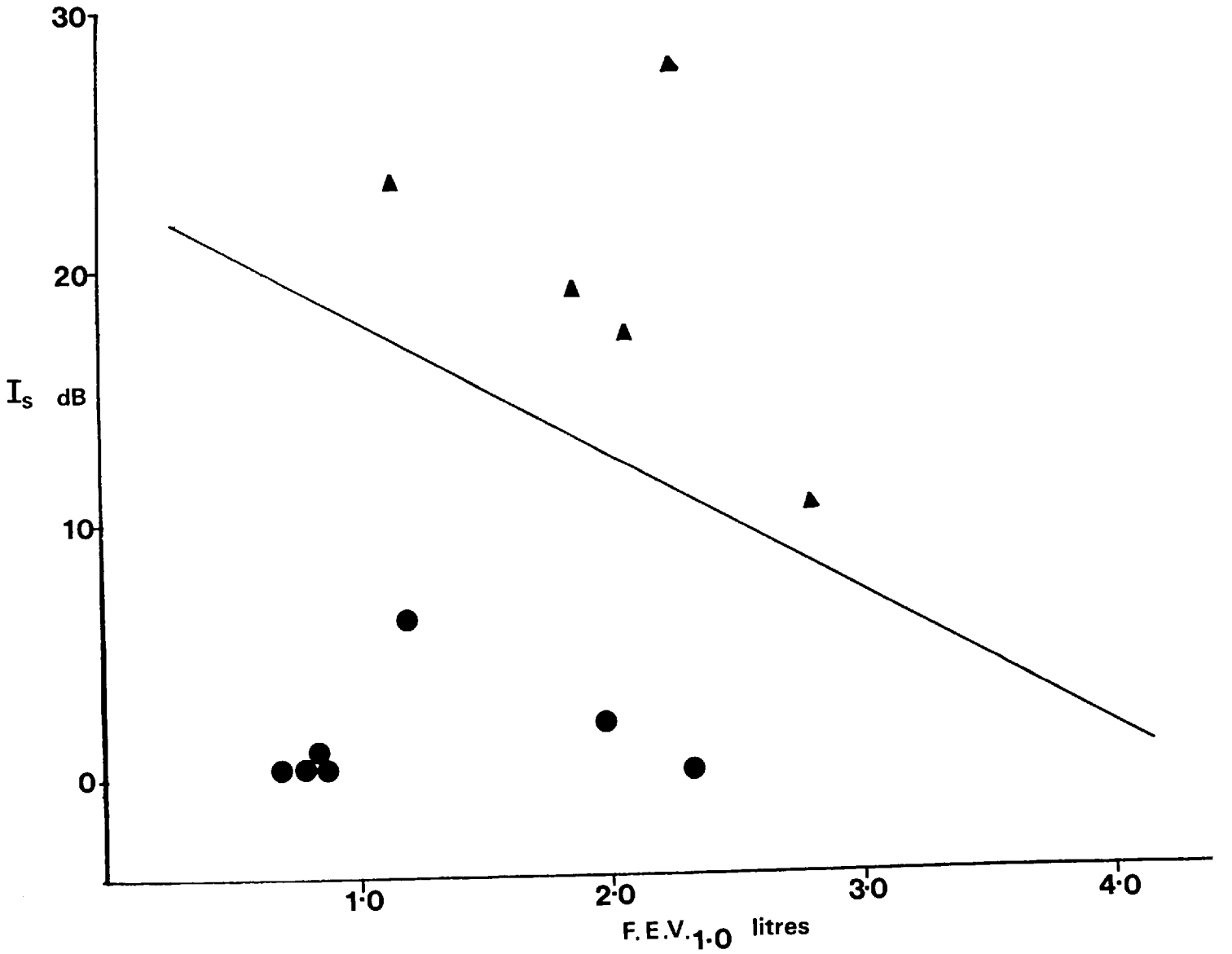


Fig 21

The inspiratory sound intensity in relation to FEV_1 in primary emphysema and in bronchial and tracheal stenosis (Regression line drawn from Fig. 13).

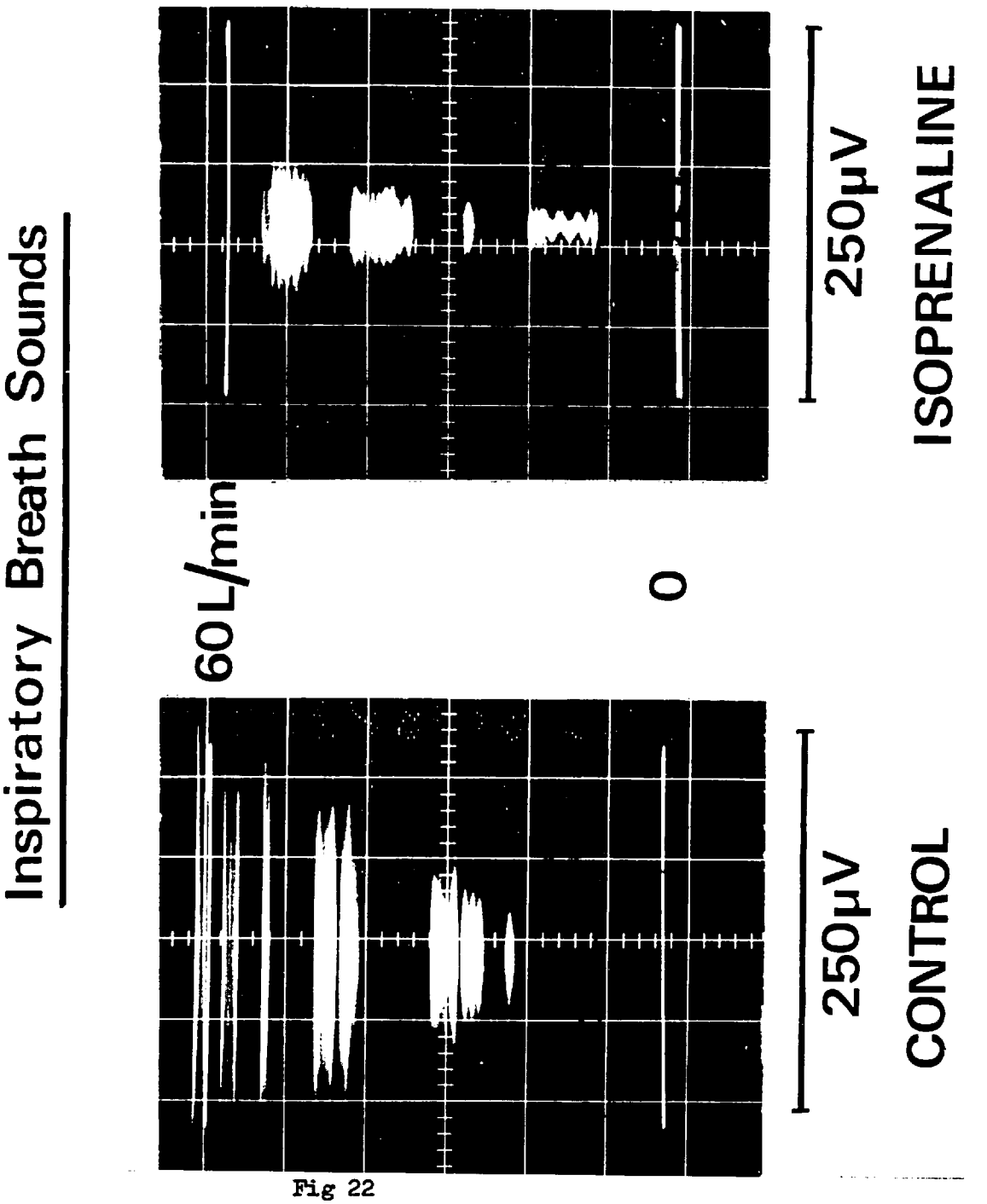


Fig 22

Decrease in inspiratory sound intensity following isoprenaline in an asthmatic subject.

before and after tracheal dilatation and the results were compared with other laboratory tests of upper airway obstruction (Table VIII).

Bronchodilator Drugs

The inhalation of isoprenaline aerosol reduced the intensity of the inspiratory breath sounds in chronic bronchitis and in asthma (Fig. 22). Observations on sixty five patients (34 with chronic bronchitis and 31 with asthma) showed a mean decrease in sound intensity of 4 dB (range 0 - 20 dB). A proportional change in the PEF, FEV_1, airway resistance, maximum mid-inspiratory flow rate, and the sound amplitude after inhalation of an isoprenaline aerosol is shown in Table IX. The magnitude of change in these measurements following inhalation of isoprenaline was approximately proportional to the changes in inspiratory sound intensity.

Atropine sulphate, 0.6 mgm was given by subcutaneous injection to 18 patients with chronic bronchitis. Measurement one hour after the injection showed a fall in inspiratory sound intensity between 1 and 19 dB (mean 5 dB). A comparison of the proportional change in PEF, FEV_1 and sound amplitude produced by atropine is shown in Table X. The magnitude of change in these measurements after atropine was approximately proportional to the changes in inspiratory sound intensity.

Table XI shows the combined effect of atropine sulphate given subcutaneously and isoprenaline aerosol administered 1 hour later on inspiratory sound intensity, PEF, and FEV_1. The changes in these measurements after atropine is expressed as a proportion of total change after atropine and isoprenaline. The change in these measurements was usually in the expected direction and of comparable magnitude but there were exceptions where the PEF and the FEV_1 improved while the inspiratory sound remained unchanged. Such a case is illustrated in Table XII. The possibility that such differences between changes in inspiratory sound intensity and the forced expiratory measurements after these drugs may

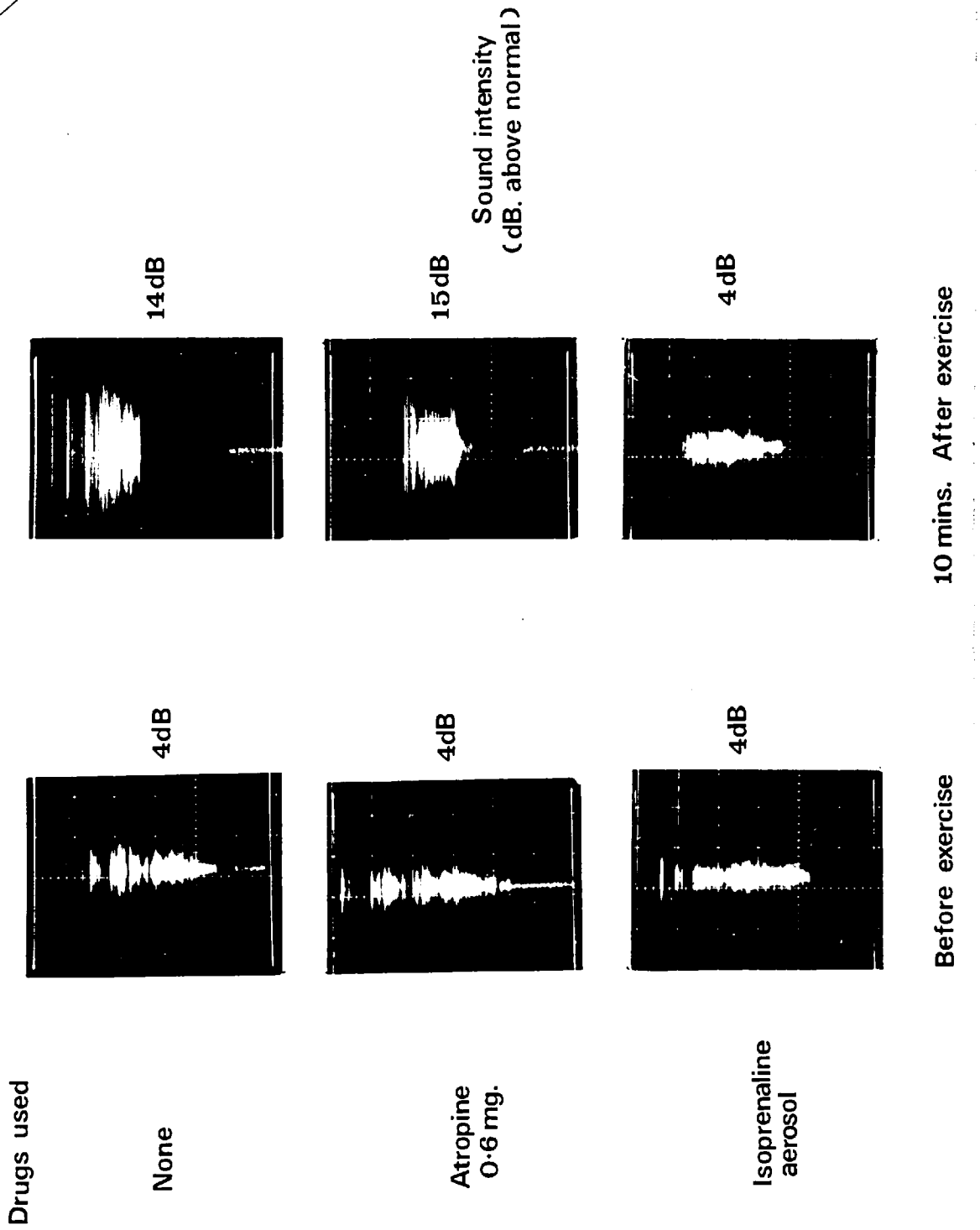


Fig 23

Prophylactic use of atropine and isoprenaline in a subject with exercise - induced asthma.

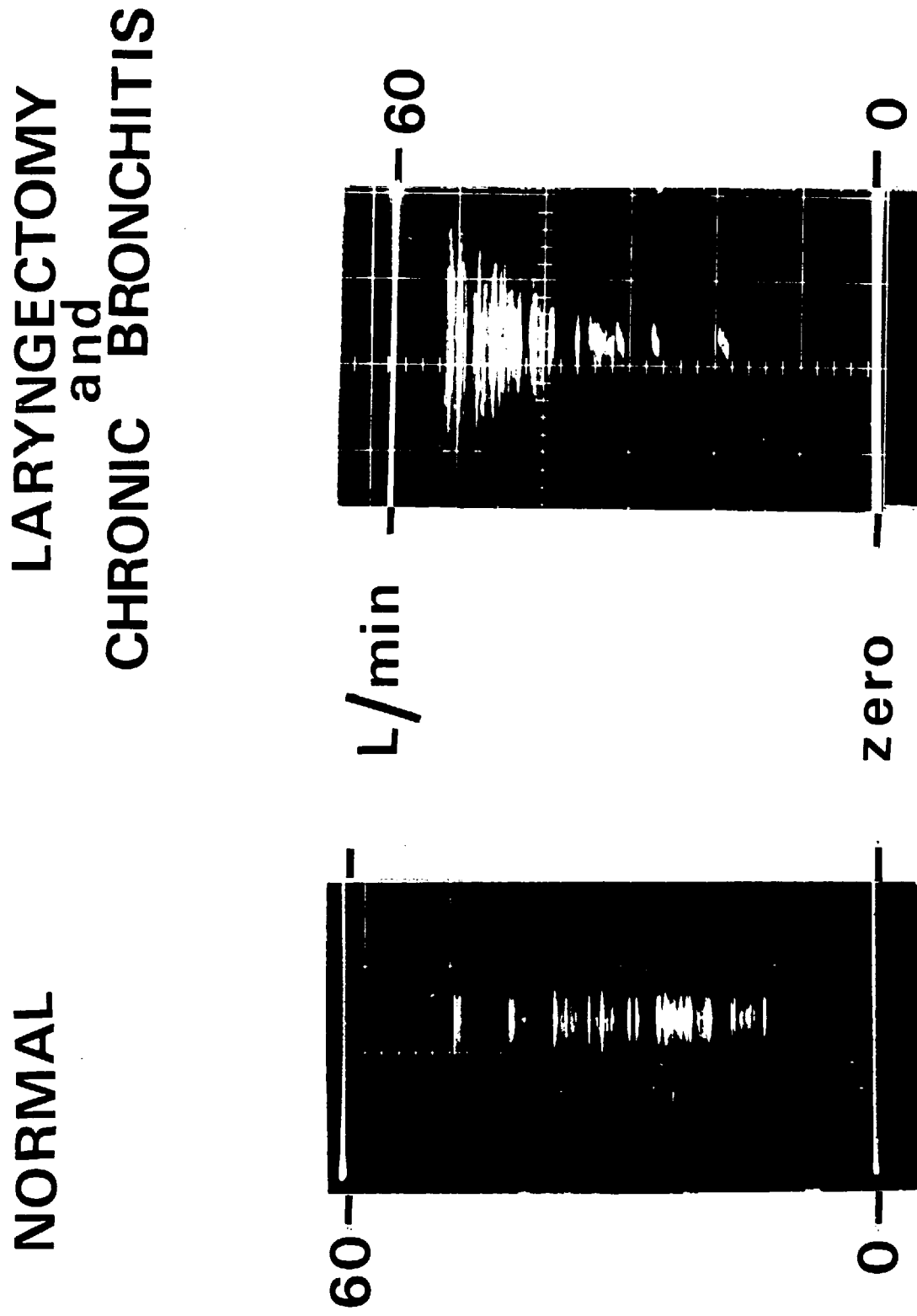


Fig 24

The inspiratory breath sounds in health and in chronic bronchitis after laryngectomy.

indicate the site of bronchodilator action is discussed later (p. 79).

Exercise Asthma

Exercise had no effect on intensity of the inspiratory sound in normal subjects. In 6 patients with exercise-induced asthma the inspiratory sound intensity rose after exercise and remained high for at least 30 mins. Isoprenaline aerosol given after exercise, reduced the inspiratory sound intensity and improved the forced expiratory tests (Table XIII).

The effect of prophylactic administration of bronchodilator drugs was studied in two of these patients. Isoprenaline aerosol given immediately before exercise prevented the rise in sound intensity, while atropine sulphate, 0.6 mgm given by subcutaneous injection one hour before exercise was ineffective (Fig. 23, Table XIV).

Laryngectomy

Sound measurements were made on six patients whose larynx had been resected for cancer some years earlier. They were breathing through a circular or oval stoma 1 - 2 cm in diameter giving a direct view into the upper 2 or 3 cm of the trachea. Sound and flow were measured by the method used on normal subjects except that the cardboard airway instead of being held between the teeth was pressed against the neck just above the jugular notch to make an airtight seal around the stoma.

All these patients had a chronic productive cough, some long before the laryngectomy, others only since the operation. Sputum was seen in the trachea and in many cases repeatedly coughed up during the examination. The FEV_1 and PEF were reduced to about half the predicted value in three patients; in the other three these could not be measured for technical reasons.

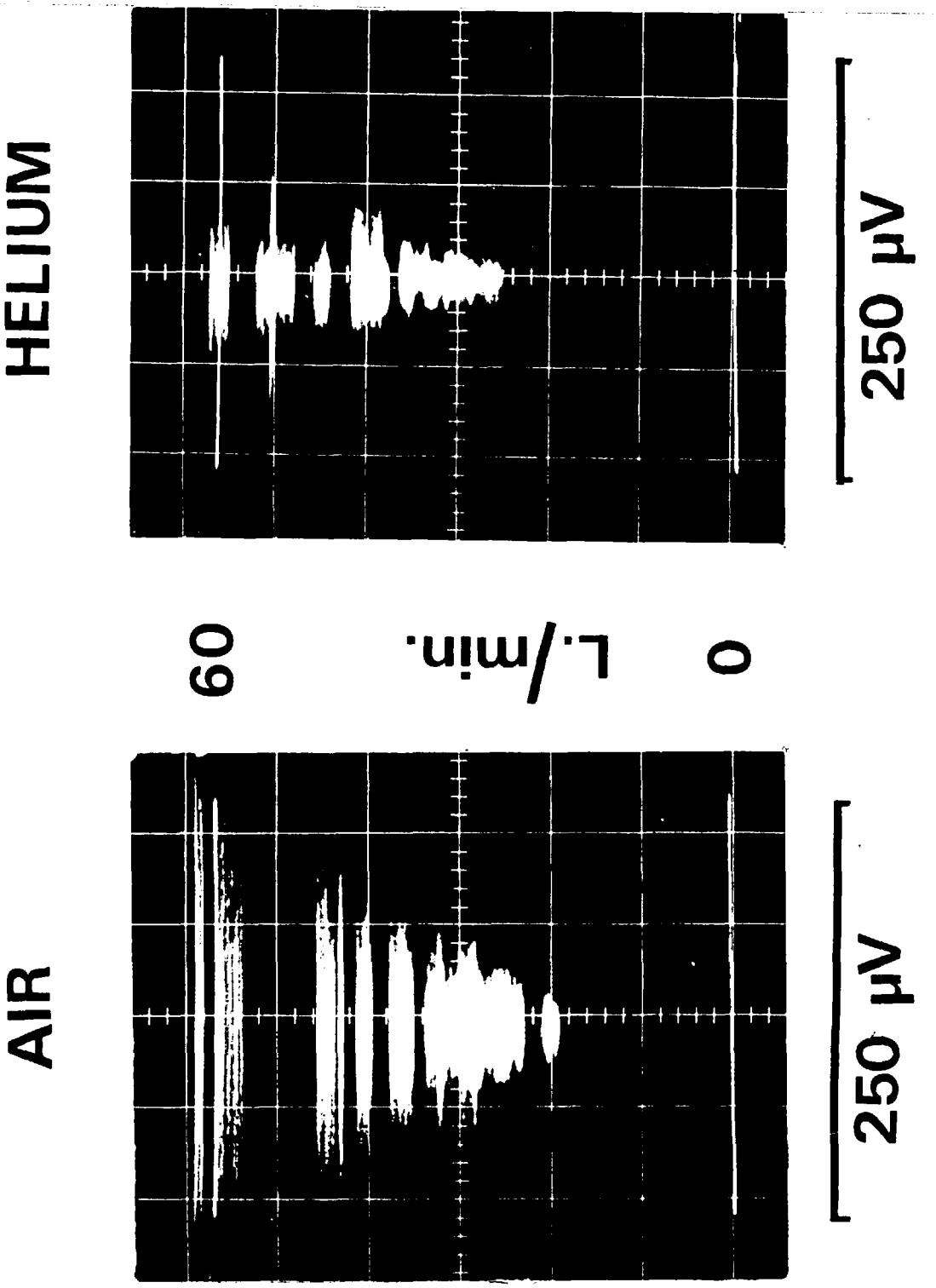


Fig 25

Decrease in inspiratory sound intensity while breathing helium in a patient with chronic bronchitis.

LARYNGECTOMY

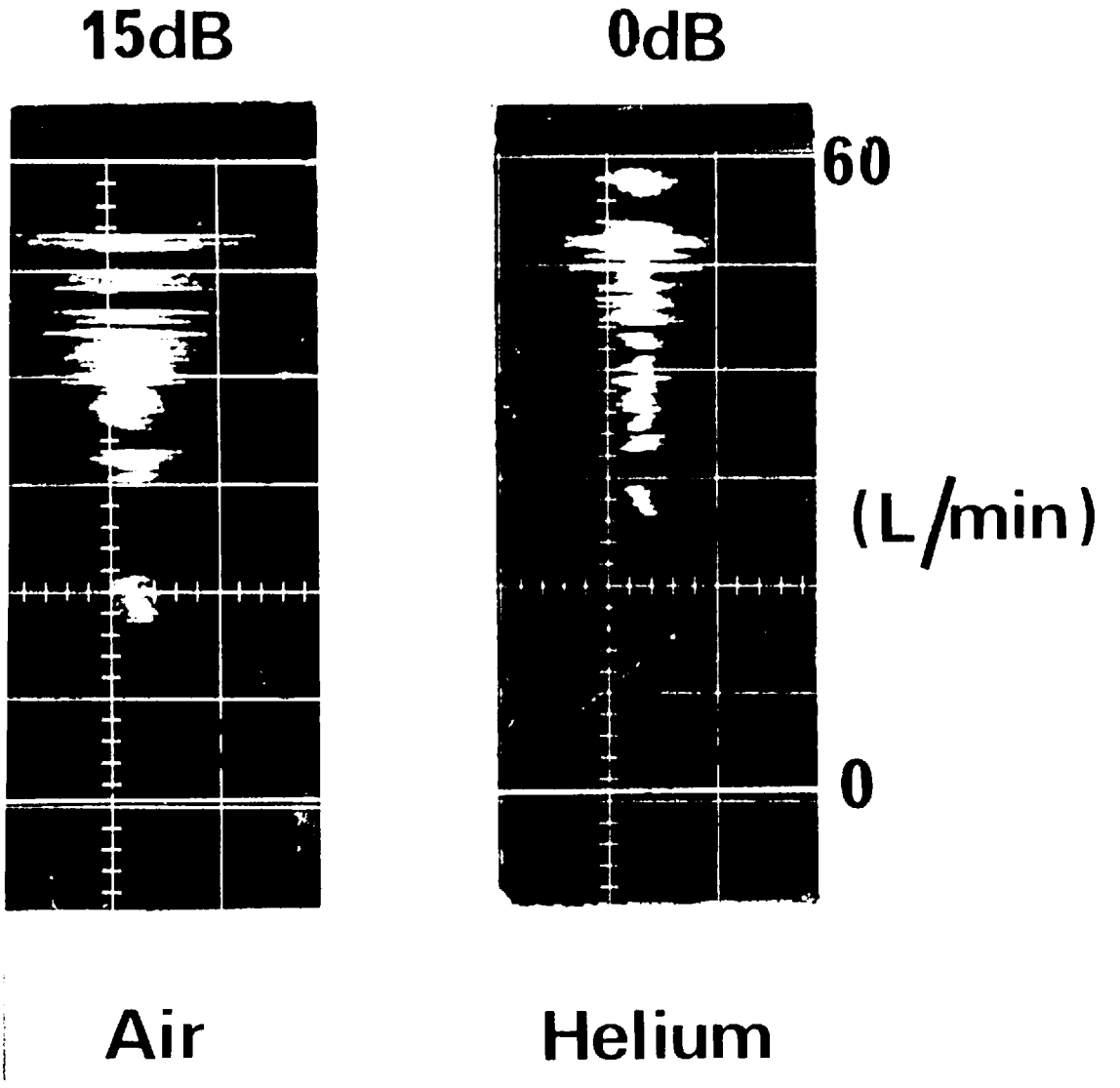


Fig 26

Decrease in inspiratory sound intensity while breathing helium after laryngectomy.

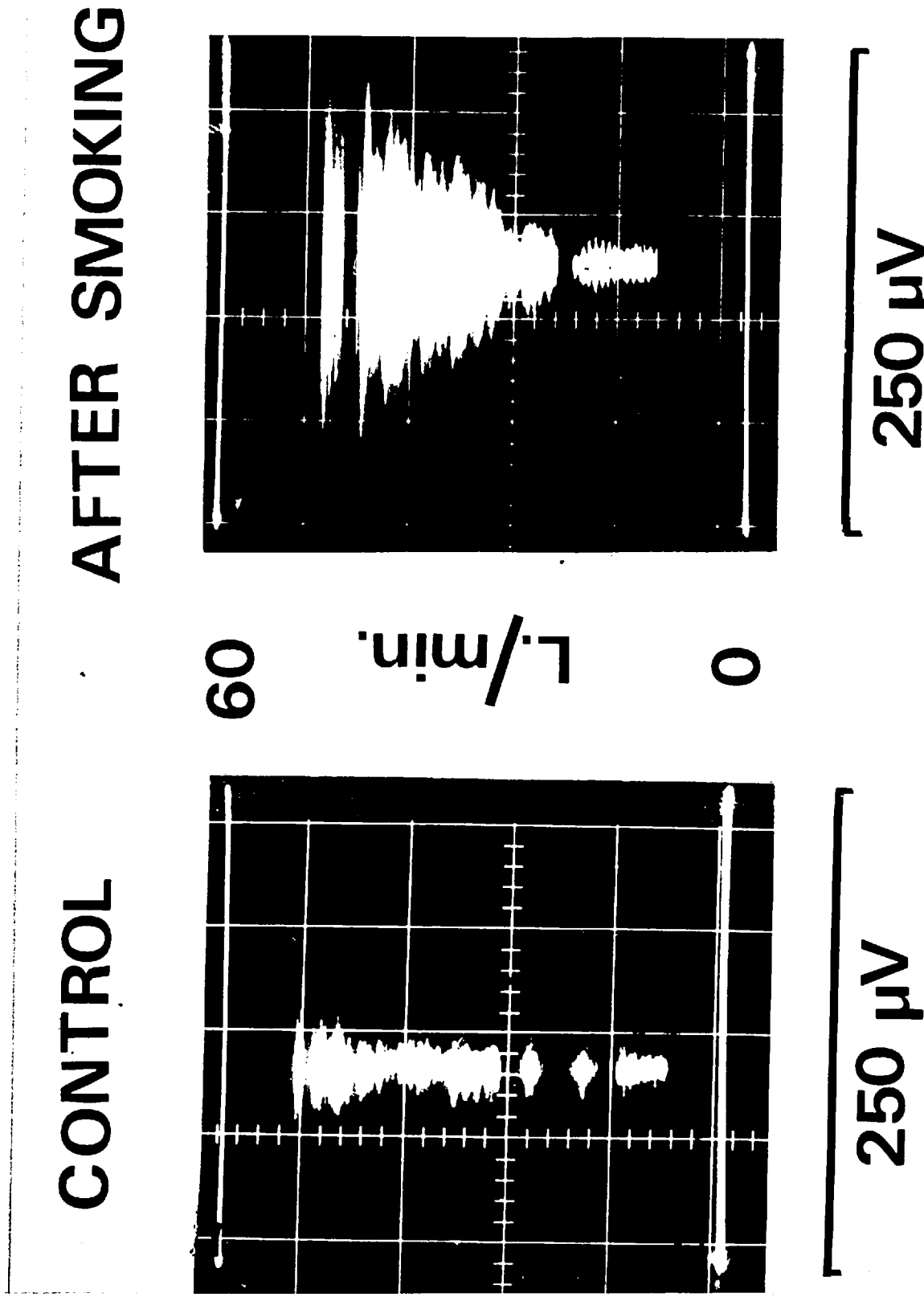


Fig 27

Rise of inspiratory sound intensity in an asthmatic subject after smoking a cigarette.

The inspiratory sound was abnormally loud in all six patients (Fig. 24, Table XV). It is not known what proportion of this noise came from the stoma, but as this orifice was much wider than the normal rima glottidis and led directly into the trachea without any abrupt change of calibre or direction its contribution to the noise was thought to be small.

Pneumonectomy

The inspiratory sound intensity was increased in three subjects studied after pneumonectomy. In two of these breath sounds were disproportionately loud for the corresponding value of PEF and the FEV_1 (Table XVI).

Helium

The effect of substituting a mixture of 79% helium and 21% oxygen for air was observed in seven patients (Figs. 25 and 26, Table XVII). In six of these patients helium produced a fall in sound intensity (Mean 9 dB, range 4 - 14 dB). In one patient the inspiratory sound remained unchanged.

Tobacco

The effect of smoking a cigarette on inspiratory sound intensity was observed in three normal and five asthmatic subjects (Table XVIII, Fig. 27). There was no significant rise in inspiratory sound intensity except in two asthmatic patients.

Breath Sounds recorded on the Chest Wall

Helium

Breath sounds were recorded on the posterior chest wall 2 cm above the diaphragm in three normal subjects. The change in sound intensity breathing air, 100% oxygen and a mixture of 79% helium and 21% oxygen was compared. In each subject breath sounds were equally loud in air and oxygen, but became quieter breathing helium (Figs. 28, 29).

Cog-wheel Respiration

Breath sounds of a subject with cogwheel breathing are illustrated in Figs. 30 and 31. The recording was obtained near the apex of the heart. The illustration shows rhythmic fall of inspiratory sound synchronous with cardiac systole. During breath holding the breath sounds are absent and only the heart sounds are seen. The significance of this observation in relation to the site of production of breath sounds is discussed later (p. 83).

Duration of breath sounds at the chest wall and phase of respirations

Fig. 32 shows the relation between the intensity of breath sounds and the volume flow rates in inspiration and expiration. Fig. 33 illustrates the possible reasons why breath sounds at the trachea and the upper chest wall are approximately of equal duration in the two phases of respiration while breath sounds at the lower regions of the chest wall are heard mainly in inspiration (p. 84).

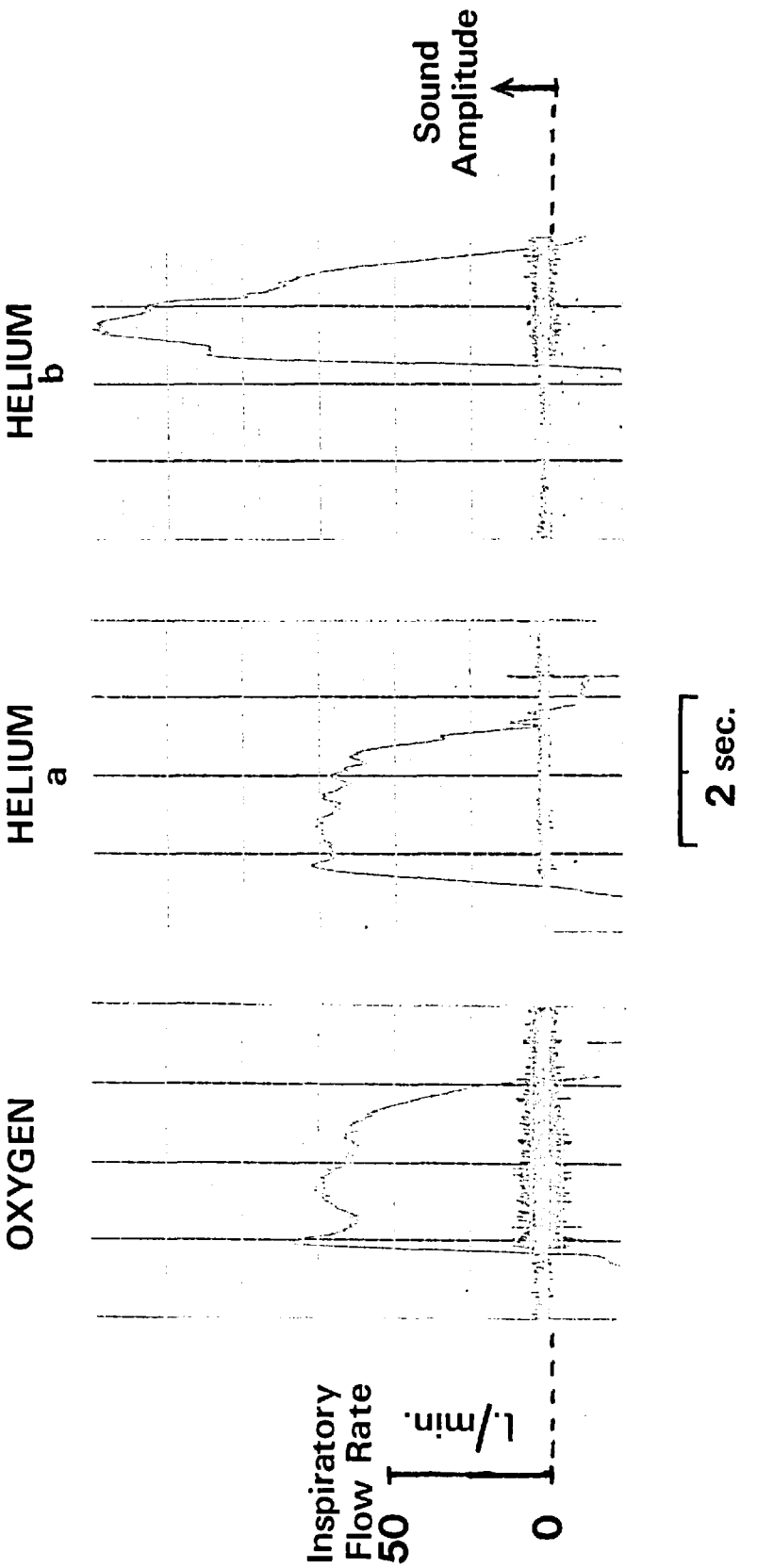


Fig 28

Breath sounds of a normal subject recorded from the chest wall. The sound amplitude decreases while breathing helium (Note that the flow rate in a is the same and in b higher than the flow rate while breathing oxygen.)

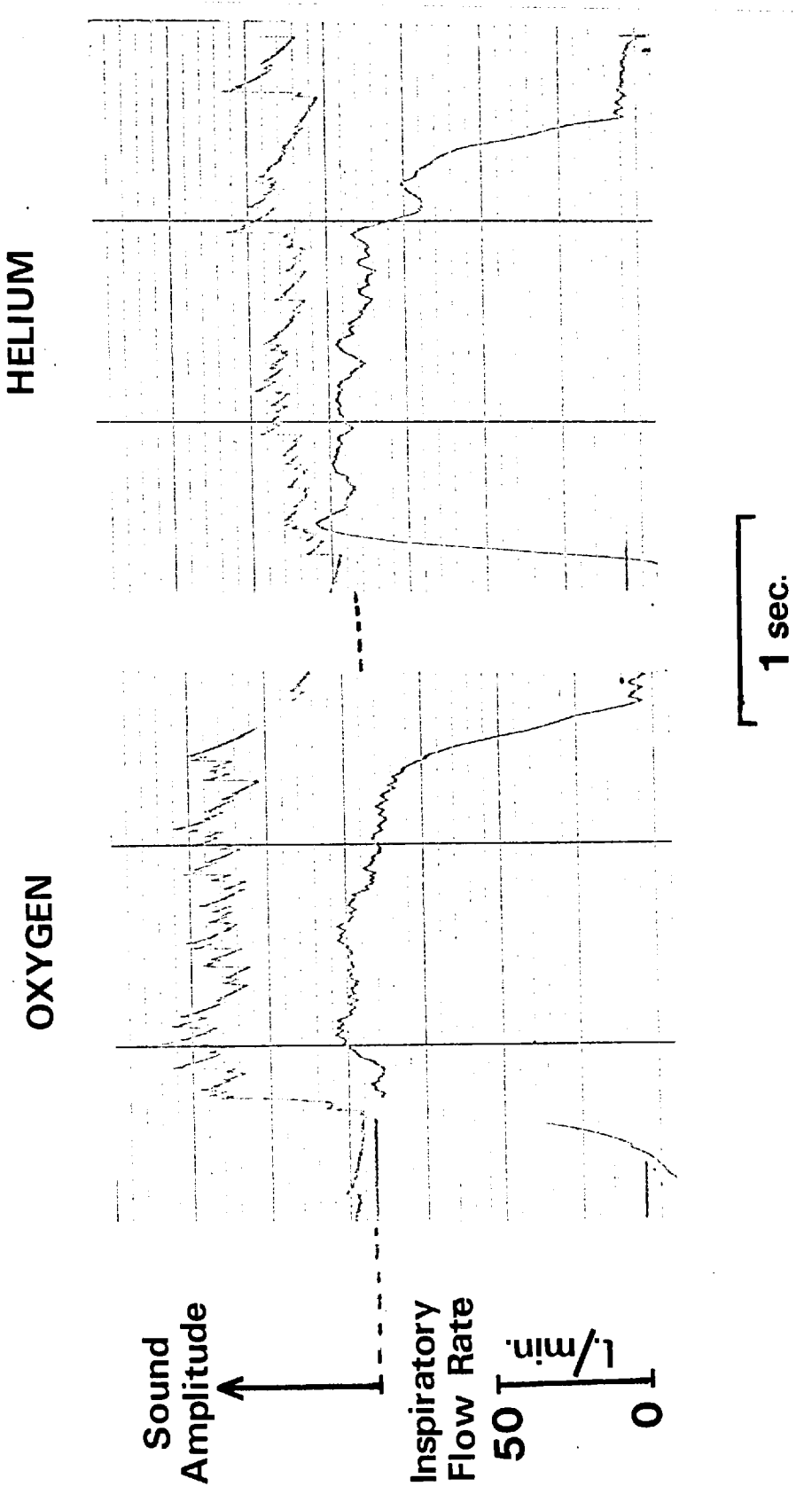


Fig 29

Breath sounds recorded from the chest wall and displayed as a rectified and filtered output of the microphone. The inspiratory sound intensity is lower with helium than with oxygen although the flow rates are indentical.

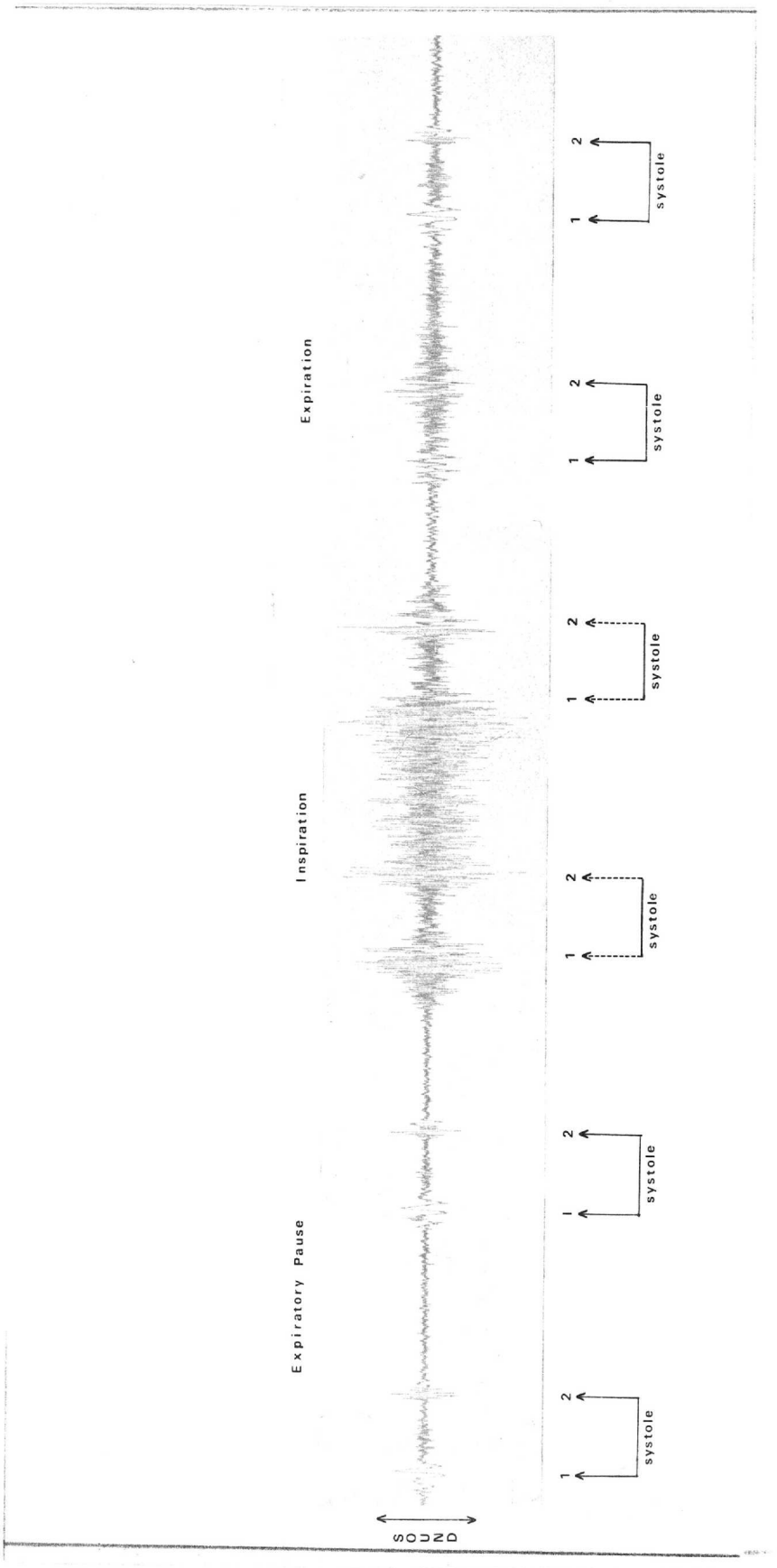


Fig 30

Breath sounds of a subject with cogwheel breathing
The inspiratory sound amplitude is reduced synchronous with cardiac systole. During breath holding only the heart sounds are seen.

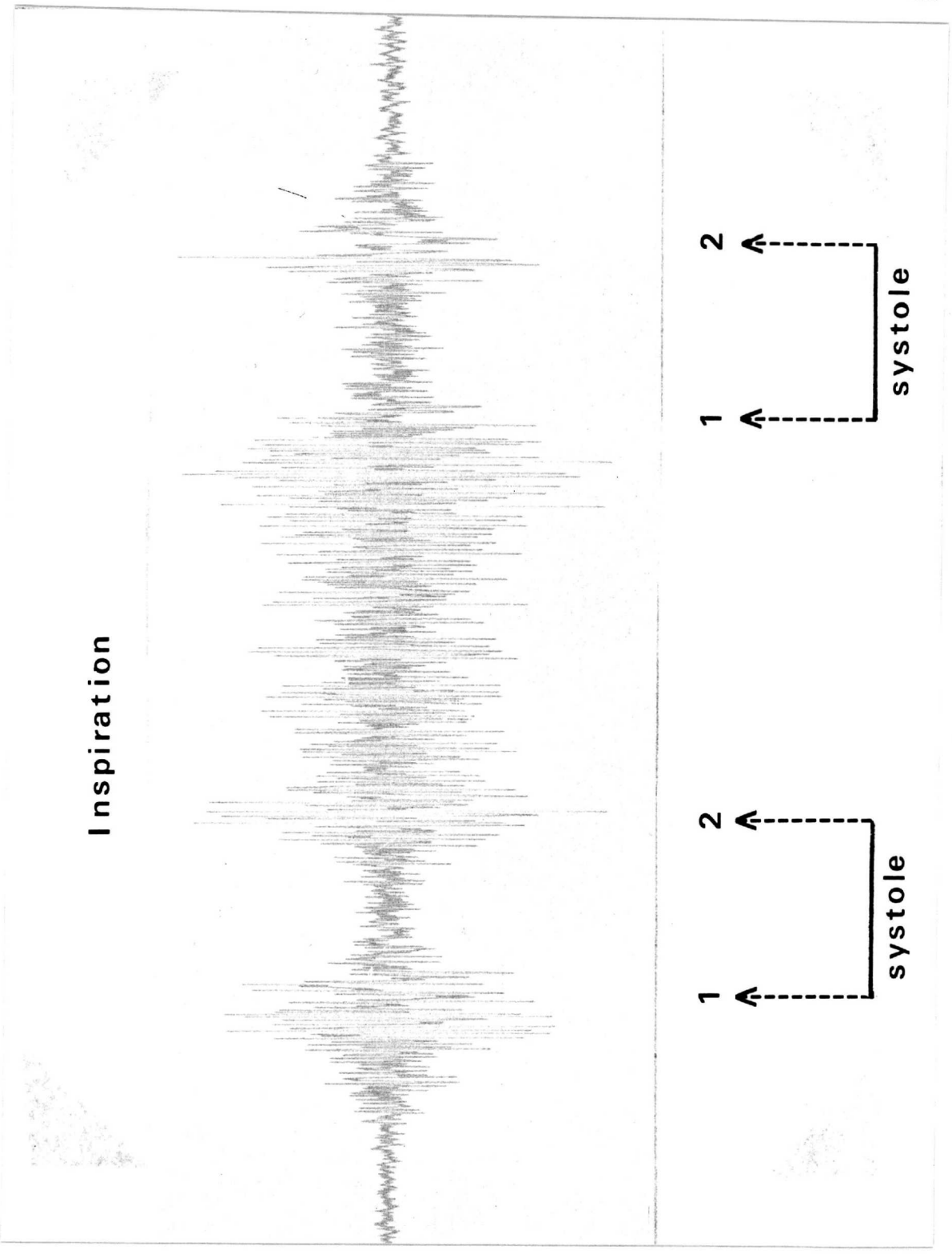


Fig 31

Close up view of inspiratory breath sounds displayed in Fig. 30.

DISCUSSION

Mechanism and site of production of breath sounds

Differing opinions as to the origin of breath sounds and their significance in diseases are largely due to uncertainty about the mechanism of sound production. Whether the breath sounds are generated by oscillations of the gas medium or the solid components of the airways may be ascertained by observing the effect of helium inhalation on the sound intensity. When helium is breathed the breath sounds become quieter indicating that they are generated by turbulent gas flow since air flow is less turbulent in gases of low density. In contrast, a wheeze has been shown to be produced by oscillations of the bronchial wall (Forgacs, 1971) and it is as loud in helium as in air because the amplitude of vibrations of the bronchial wall is not affected by the ambient gas.

Breath sounds are produced when part of the kinetic energy of airflow is converted into sound. The site of production of breath sounds may be inferred from flow conditions within the airways. Although the flow pattern in human airways is complex, it is convenient to consider it in terms of laminar and turbulent airflow in a rigid pipe of uniform calibre. When a streamline flow is maintained, Pouiseuille calculated that the volume rate of airflow is directly proportional to the driving pressure and the fourth power of the radius of the tube, and inversely proportional to its length. The transition from laminar to turbulent airflow is predicted from Reynolds' number (Re) calculated from dimensions of the pipe, density and the viscosity of gas. ** The critical value of Re has been found to be 2000, when laminar flow changes to turbulent flow (Jaeger and Matthys, 1970).

** $Re = \frac{D \dot{V} d}{A.u}$ where D = diameter, $\dot{V}$ = volume flow rate, d = gas density, A = cross-sectional area and u = gas viscosity.

The equation for determining turbulent airflow is only an approximate statement when applied to human airways. The mouth, pharynx, larynx and the trachea are tubes of irregular calibre in series. Turbulence in these airways is produced at a lower Reynolds' number than in a smooth pipe of uniform calibre. The bronchial tree is a complex system of branching and rebranching tubes in parallel. The large increase in the number of airways conducting air in parallel outweighs the effect of their decreasing calibre. The result is a considerable expansion of the total cross-section of the airways with a corresponding fall in the volume rate of gas flow from proximal bronchi towards the periphery of the lung.

The predictions based on Reynolds' number suggest that at all but the lowest inspiratory flow rates turbulence occurs in the upper airways, trachea and the main bronchi. It may persist in lobar bronchi since turbulent eddies need a certain distance to decay. The airflow in succeeding generations of airways extending up to about the tenth generation counting from the trachea is laminar but disturbed (Pedley et al, 1971). The disturbance occurs at branching points due to abrupt change in the direction of airflow. In the airways beyond the tenth generation there is a progressive fall in the volume flow rate and the airflow more closely approximates true laminar flow.

Breath sounds are likely to be generated only within those airways where the flow conditions are appropriate for oscillations of gas to occur. The random movements of gas molecules in regions of turbulent airflow particularly favour sound production. An additional source of sound may be expected in the transitional zone of airways where abrupt changes in the direction of airflow at the branching sites result in formation of turbulent eddies. However the slow flow velocities at physiological rates of breathing in the peripheral airways and the alveoli is incompatible with sound production in a gas phase.

The source of breath sounds therefore include the upper airways, the trachea and the proximal intrathoracic airways up to about the tenth generation. In the airways beyond the tenth generation it is improbable that breath sounds can be produced in a gas phase. Further evidence in support of this view is discussed later.

Breath sounds at the mouth in diffuse airway obstruction and stenosis of the upper airways

Loud breath sounds heard at the mouth in patients with chronic bronchitis and asthma are commonly thought to originate in the pharynx and the larynx. The observations on inspiratory sound intensity in these patients cannot be explained on this belief unless an improbable assumption is made that the patients with diffuse airway obstruction in some way alter the shape of the pharynx and the position of the vocal cords in a manner different from normal subjects during quiet inspiration.

Good inverse correlation between the inspiratory sound intensity and the forced expiratory tests in patients with chronic bronchitis and asthma suggest that noisy breathing in these patients is closely related to calibre changes of the intrathoracic airways. This view is supported by the observation that bronchodilator drugs reduced the sound intensity when this was increased and the finding that exercise is some asthmatic subjects caused a large increase in inspiratory sound intensity while isoprenaline inhalation prevented a rise after exercise. The close relation between the intensity of inspiratory breath sounds and the calibre of the bronchi may be inferred from the observation that in subjects with cogwheel breathing the sound intensity fluctuated rhythmically with cardiac systole. It is known that ventricular systole is accompanied by a transient fall in intrathoracic pressure (Mills, 1969) which will tend to widen the intrathoracic airways. In this situation the rhythmic fall of inspiratory breath sound intensity is explained by a fall in linear velocity of airflow in the lobar and segmental bronchi of the left lower lobe during cardiac systole.

Strong supporting evidence for the suggestion that the pharynx and the larynx contribute little to loud breath sounds in patients with chronic bronchitis and asthma comes from the observation that the intensity of inspiratory breath sounds is abnormally loud in patients with chronic bronchitis who have undergone laryngectomy. It falls short of conclusive proof only because it is not known what proportion of the inspiratory sound was produced at the stoma. As the stoma was often as wide as the trachea itself it seems reasonable to conclude that loud breath sounds in laryngectomy patients originate in the trachea and the intrathoracic airways.

Increased turbulence responsible for loud breath sounds may in principle be due to surface irregularities in the airways, abrupt changes in the direction of flow, or to narrowing of airways resulting in more rapid flow. All these factors may come into play occasionally in disease, but the large fall in sound intensity produced by bronchodilator drugs and the rise following exercise provocation tests show that turbulence in chronic bronchitis and asthma is closely related to the calibre of the airways.

The limitations to expiratory flow rate depend upon several factors which operate during forced expiration. These include the volume and elastic recoil of the lungs, collapsibility of the flow limiting airways and resistance of the upstream airways. Although the relative contributions of these factors in limiting the expiratory flow remain to be defined, it is believed that in chronic bronchitis and asthma, high intrapulmonary resistance with some loss of elastic recoil are the more important determinants (Pride, 1971). The good inverse correlation between the inspiratory sound intensity and the forced expiratory tests in these patients suggests that intrathoracic airways up to the tenth generation provided an important source of loud breath sounds. In these circumstances premature expiratory compression of the large airways indicated by the low FEV_1 and PEF may be explained by a steep fall in intrabronchial pressure as a result of high flow resistance between alveoli and the main bronchi.

When the inspiratory sound measurements and the forced expiratory tests in primary emphysema were compared with those of patients with chronic bronchitis and asthma an important difference was seen. In primary emphysema, in contrast to bronchitis and asthma, the intensity of inspiratory breath sounds is normal in the face of the FEV_1 and PEF which are often very low. In five patients with this combination of findings the inspiratory airway resistance was normal while in one patient it was raised. The normal inspiratory sound intensity and the usually normal inspiratory airway resistance indicate that inspiratory calibre of the airways in this disease is not greatly reduced. The severe reduction in expiratory flow is explained by premature dynamic expiratory collapse of the large bronchi, in part due to the loss of elastic recoil of the lung (Mead et al, 1967; Black et al, 1972).

The breath sounds in bronchial and tracheal stenosis were observed to be disproportionately loud. The loud breath sounds in these patients contrast with the quiet breathing in primary emphysema. The increased inspiratory sound intensity in patients with bronchial stenosis may either be due to increase in the volume rate of airflow in the patent bronchi or to an increase in linear velocity of airflow at the site of stenosis. An example of the first mechanism is the loud breath sounds in patients with pneumonectomy. The second mechanism however, is probably the main cause of the increase in sound intensity. Supporting this Bullar (1884) experimenting on excised calf lungs noted loud breath sounds when a bronchus was compressed, and in a patient with cicatricial tracheal stenosis the increased inspiratory sound intensity was reduced after tracheal dilatation.

Comparison of the inspiratory sound intensity with the FEV_1 may help to indicate the nature of the airways obstruction. A normal inspiratory sound intensity in presence of a low FEV_1 suggests calibre changes of the peripheral airways while loud inspiratory breath sounds and a normal or

slightly reduced FEV_1 point to narrowing of the larger airways. The inspiratory sound intensity compared to the other tests of upper airways obstruction show that it is a sensitive measurement and can be included in the assessment of laryngeal and tracheal stenosis.

In chronic bronchitis the vital capacity correlated inversely with the inspiratory sound intensity. In asthmatic patients, however, a poor correlation resulted because the breath sounds of some patients were louder in relation to the vital capacity than was the case in chronic bronchitis (Figs. 15, 16). In asthma there are regional variations in the degree of narrowing of the airways (Bentivoglio et al, 1963). The loud inspiratory sounds in some asthmatics with normal or slightly reduced vital capacity may have been due to narrowing of one or two large bronchi. The observations on patients with stenosis of one of the main bronchi show that loud breath sounds may be of focal origin. Thus the intensity of inspiratory breath sounds in such cases would fail to reflect conditions of the airways in general.

The measurements of the inspiratory airway resistance by body plethysmography would be expected to correlate well with the inspiratory sound intensity since airway resistance measured in this way is believed to be influenced mainly by the upper airways. Although a statistically significant correlation was found between the inspiratory sound intensity and airway resistance in chronic bronchitis, the correlation was not as good as with the FEV_1 and the PEF. The correlation was not further improved when the inspiratory sound intensity was compared with the specific airway resistance or with the logarithm of airway resistance and the logarithm of specific airway resistance.

The influence of lung volume may have contributed to poor correlation between the inspiratory sound intensity and the airway resistance. Changes in lung volume may cause large changes in airway resistance. The inspiratory resistance rises as the lungs are deflated below functional

residual capacity and falls when the lungs are inflated above functional residual capacity. The inspiratory breath sounds were recorded during quiet breathing while airway resistance was measured at a lung volume selected by the patient to perform panting manoeuvre. Sterling (1967) pointed out that the 'spontaneous' intrathoracic gas volume so measured is usually higher than the functional residual capacity. During panting there is a rapid increase in lung volume for a few breaths after which the lung volume is relatively steady for a time although further fluctuations may occur if panting is prolonged. Although the airway resistance was likely to have been measured at a lung volume higher than the functional residual capacity, examination of Fig. 19 and the lack of improvement in correlation between the inspiratory sound intensity and the specific airway resistance suggests that differences in the lung volume between the two measurements is unlikely to have been of major importance.

Observation on patients with localised bronchial and tracheal stenosis showed that the inspiratory breath sounds in these patients were usually disproportionately loud. In some patients with chronic bronchitis and asthma focal narrowing of one or two large airways may have caused a large increase in inspiratory sound intensity. Such regional variations in airway calibre, however, would not be expected to cause a corresponding change in total airway resistance as measured by the body plethysmograph. This view is supported by the finding (Fig. 19) that in some patients inspiratory sound intensity was increased when the airway resistance was normal but the reverse relationship between the two measurements was not encountered in these patients.

Precise reasons why there is a poor correlation between the inspiratory sound intensity and airway resistance in patients with chronic bronchitis and asthma remain uncertain. However, as might be anticipated, a close relationship between the two measurements was maintained in patients

with primary emphysema and tracheal stenosis. In primary emphysema the inspiratory sound intensity and the inspiratory airway resistance were usually normal while in tracheal stenosis both measurements were increased.

During forced inspiration, resistance to gas flow increases with increasing turbulence. In patients with chronic bronchitis and asthma turbulence is further intensified in the narrowed intrathoracic airways. The maximum mid-inspiratory flow rate and the inspiratory sound intensity as measured in this study depend upon airway calibre at about the same lung volume. A significant inverse correlation between the two measurements in chronic bronchitis and asthma is therefore not unexpected.

Isoprenaline and atropine reduced the inspiratory sound intensity. In general the magnitude of the reduction in sound intensity was of the same order as the improvement in forced expiratory tests, airway resistance and the maximum mid-inspiratory flow rate. The site of action of a drug may be inferred from comparison of the inspiratory sound intensity and the forced expiratory tests. Although isoprenaline and atropine are not known to have a selective action on either the peripheral or central bronchi in one patient with diffuse airway obstruction the PEF and the FEV_1 improved after administration of these drugs while the inspiratory sound intensity remained unchanged. The reason for this difference is either fixed stenosis of a single large airway or selective action of the drug on peripheral airways which make little contribution to sound. Conclusions on similar lines may be drawn on the site of action of a drug when the inspiratory sound intensity and the forced expiratory measurements do not change in the expected direction after the administration of a bronchodilator or a bronchoconstrictor drug.

Altounyan (1964) suggested that patients with non-allergic airway obstruction, or patients with allergic airway obstruction adequately controlled on corticosteroids have a good response to atropine as measured

by the FEV_1 compared with the subsequent response to isoprenaline. By contrast patients who subsequently respond to corticosteroids show little or no response to atropine compared to their response to isoprenaline. The observations on patients with chronic bronchitis showed that the change in inspiratory sound intensity was comparable to FEV_1 after administration of atropine and subsequent isoprenaline. The sound intensity measurement may therefore be used in assessment of corticosteroid responsiveness in patients with airways obstruction.

Exercise asthma and Sound intensity

Exercise provocation in five asthmatic subjects resulted in a large increase in the inspiratory sound intensity and a fall in the FEV_1. Isoprenaline aerosol subsequently administered reduced the inspiratory sound intensity and increased the FEV_1. In two additional asthmatic patients with a history of exercise induced asthma changes in the inspiratory sound intensity and the FEV_1 were compared so as to test the prophylactic effect of atropine and isoprenaline. The relative changes in the two measurements were similar. It is of interest that an isoprenaline aerosol blocked the effect of exercise provocation while atropine failed to do so in both subjects.

Tobacco smoke and Sound intensity

The inhalation of tobacco smoke may cause widespread narrowing of the airways and increase airway resistance (Sterling, 1967; Guyatt et al, 1970). It seemed possible that the inspiratory sound intensity might provide a more sensitive index of minor calibre changes in the large airways and thus help to identify susceptible individuals. Both of two asthmatic subjects studied in this group showed significant changes both in sound intensity and the FEV_1 reversed by isoprenaline while five normal subjects showed no change.

Bedside diagnosis

Observations on the inspiratory sound intensity can be helpful at the bedside. The changes in sound intensity at source is conveniently elicited by listening to breath sounds at the mouth with the unaided ear. The breathing of a healthy subject is silent or barely audible at the mouth. Patients with diffuse airway obstruction due to bronchitis and to asthma commonly have loud breath sounds, sometimes audible across the room. Skill in the clinical judgement of loudness of breath sounds in relation to the rate and depth of breathing is readily acquired. As a rule, the louder the breath sounds the more severe the airways obstruction. In airways obstruction of mild or moderate severity loud breathing is commonly present in the absence of a wheeze, and is perhaps a more constant auscultatory sign of airways obstruction. In pure emphysema inspiratory breath sounds at the mouth are normal because the sound intensity at source is normal, while loud breath sounds in the absence of airways obstruction as indicated by normal spirometry or forced expiratory time suggest stenosis of the larynx, trachea or the main bronchus.

Breath Sounds heard at the Chest Wall

In the preceding discussion it was suggested that the site of production of breath sounds include the upper airways, trachea and the proximal intrathoracic airways up to about the tenth generation. It is, however, commonly believed that there is an additional source of breath sounds in the terminal bronchioles and the alveoli.

The belief in the peripheral source of breath sounds stems from the observation that two varieties of breath sounds are recognized on auscultation of the chest. Laennec (1819) named these the "bronchial" and the "vesicular" respiration. An important difference between the two sounds is that the former is heard in both phases of respiration while the latter is confined to inspiration, the expiratory component when present is heard only in early expiration. Laennec thought that the "vesicular" breath sounds

were produced by friction of air with the walls of the terminal bronchioles and the alveoli. Martini (1921) on the other hand suggested that the sound was produced in inspiration by oscillations of solid components of the alveoli. Beau (1834), however, believed that breath sounds heard through the chest wall were transmitted from pharynx while Pratt and Bushnell (1925) attributed their origin to vibrations of the vocal cords.

There are two possible mechanisms generating the breath sounds heard through the chest wall. Either they are produced by oscillations of the gas or of solid components of the airways. The intensity of breath sounds generated in a gas phase would be expected to be lower in gases of low density where airflow is less turbulent. The observed fall in sound intensity during helium inhalation (Figs. 28, 29) indicates that breath sounds heard through the chest wall are generated in the gas medium. An alternative explanation based on an improbable assumption is that breath sounds were produced by oscillations of the bronchial wall and they were transmitted less effectively through helium containing lung.

For a given inspiratory flow rate, the mean velocity of gas stream in individual bronchi decreases with each generation as the total cross-sectional area increases towards the periphery. The flow conditions in the airways correspondingly change from proximal to distal bronchi. At physiological rates of inspiration, the airflow is turbulent in the upper airways, trachea and the main bronchi. In the succeeding airways up to about the tenth generation, the airflow although non-turbulent remains disturbed because of abrupt changes in the direction of airflow at the site of each branching (Pedley et al, 1971). However, the airflow in the peripheral airways and the alveoli is laminar at all physiological rates of inspiration (Jaeger and Matthys, 1970).

The oscillations of a gas medium required to generate breath sounds is possible only within those airways where airflow is non-laminar. The turbulent eddies generated in the larger airways and at branching sites in airways up to the tenth generation can be expected to be the main source of breath sounds. The sluggish rate of gas flow in the peripheral airways and the alveoli is incompatible with sound production in a gas phase. The inference is therefore that the breath sounds heard through the chest wall are transmitted from proximal bronchi.

The heart beat is known to alter flow conditions in different regions of the lung (West and Hugh-Jones, 1961). The effect of ventricular systole on the lung is two-fold. First, ventricular systole is accompanied by a transient fall in intrathoracic pressure (Mills, 1969). Second, in ventricular systole there is a sudden linear displacement of the heart away from the left lower lobe with consequent abrupt expansion of the adjoining pulmonary alveoli (Palmieri, 1962; Palmieri et al, 1962). The result of these two mechanisms operating in the left lower lobe is that during inspiration volume flow rate is augmented in the lobar bronchus synchronously with cardiac systole. West and Hugh-Jones (1961) showed that the magnitude of the volume rate of gas flow in the lobar and segmental bronchi caused by the heart beat can be large and may reach up to a third of inspiratory volume flow rate. This pulsatile gas flow continues during breath holding when a large proportion of gas moving into the left lower lobe is presumably directed to abruptly expanded alveoli in the vicinity of the heart.

Comparison of the velocity of gas flow in the terminal airways and the alveoli during inspiration and breath holding in subjects with marked cardiac pulsations is of particular interest. In inspiration the mean velocity of gas flow becomes progressively slower towards the periphery because of increase in the total cross-sectional area of the smaller airways.

Thus at an inspiratory flow rate of 50 l/min., the velocity of gas flow in the terminal airways and the alveoli is quite small and is of the order of 5 mm/sec. The velocity of gas flow in the peripheral airways of the left lower lobe during breath holding is not known. However, since the volume rate of cardiogenic gas flow in the lobar and segmental bronchi is large and mostly directed into the region of the left lower lobe adjoining the heart, it is probable that the velocity of gas flow in abruptly distended alveoli adjacent to the heart is at least of the same order of magnitude as that during inspiration. If breath sounds were generated by slow rate of inspiratory flow into the peripheral airways and the alveoli, then during breath holding breath sounds should also be audible in the region of the apex of the heart. In reality breath sounds are absent during breath holding even in subjects with cogwheel breathing who show forceful cardiac pulsations (Figs. 30, 31).

The source of breath sounds transmitted through the lung are the upper airways, trachea and the proximal bronchi. The observations of Nairn and Turner-Warwick (1969) and Leblanc et al (1970) that the intensity of breath sounds on the chest wall correlate well with regional distribution of ventilation suggest that the major sound contribution comes from the lobar and segmental bronchi supplying the lung under the stethoscope.

The difference in the duration of breath sounds in the two phases of respiration has not previously been adequately explained. Breath sounds at the mouth, trachea and the main bronchi are heard in both phases of respiration whereas breath sounds on the lower regions of the chest wall are usually heard only in inspiration, an expiratory component when present is confined to early expiration.

The intensity of breath sounds is flow rate dependent and closely follows the shape of the inspiratory and expiratory flow rate curves. The inspiratory flow curve is identified by three phases: a brief

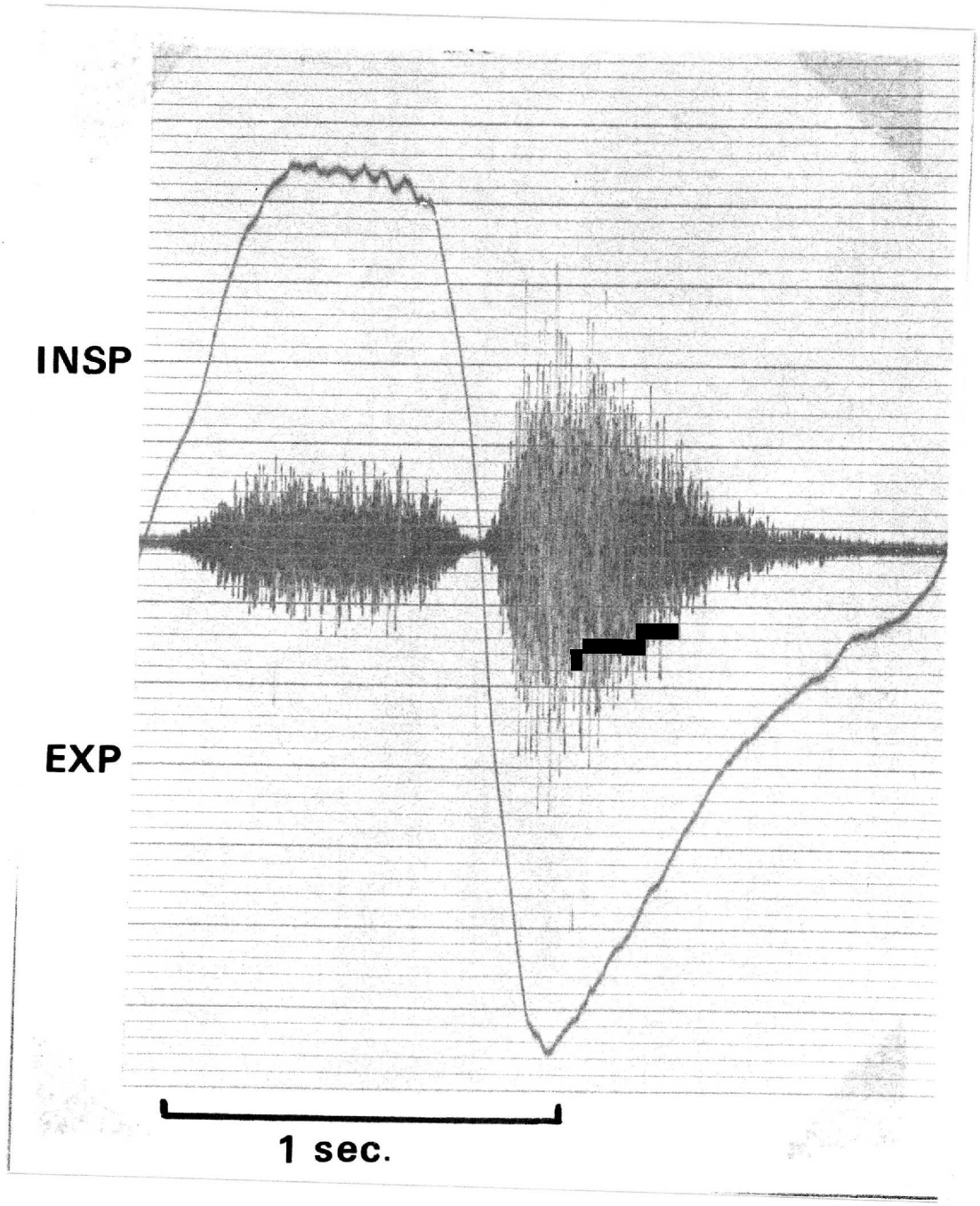


Fig 32

Breath sounds related to the shape of the inspiratory and the expiratory flow rate curves. The sound amplitude is maximum in early expiration but approximately evenly distributed in inspiration.

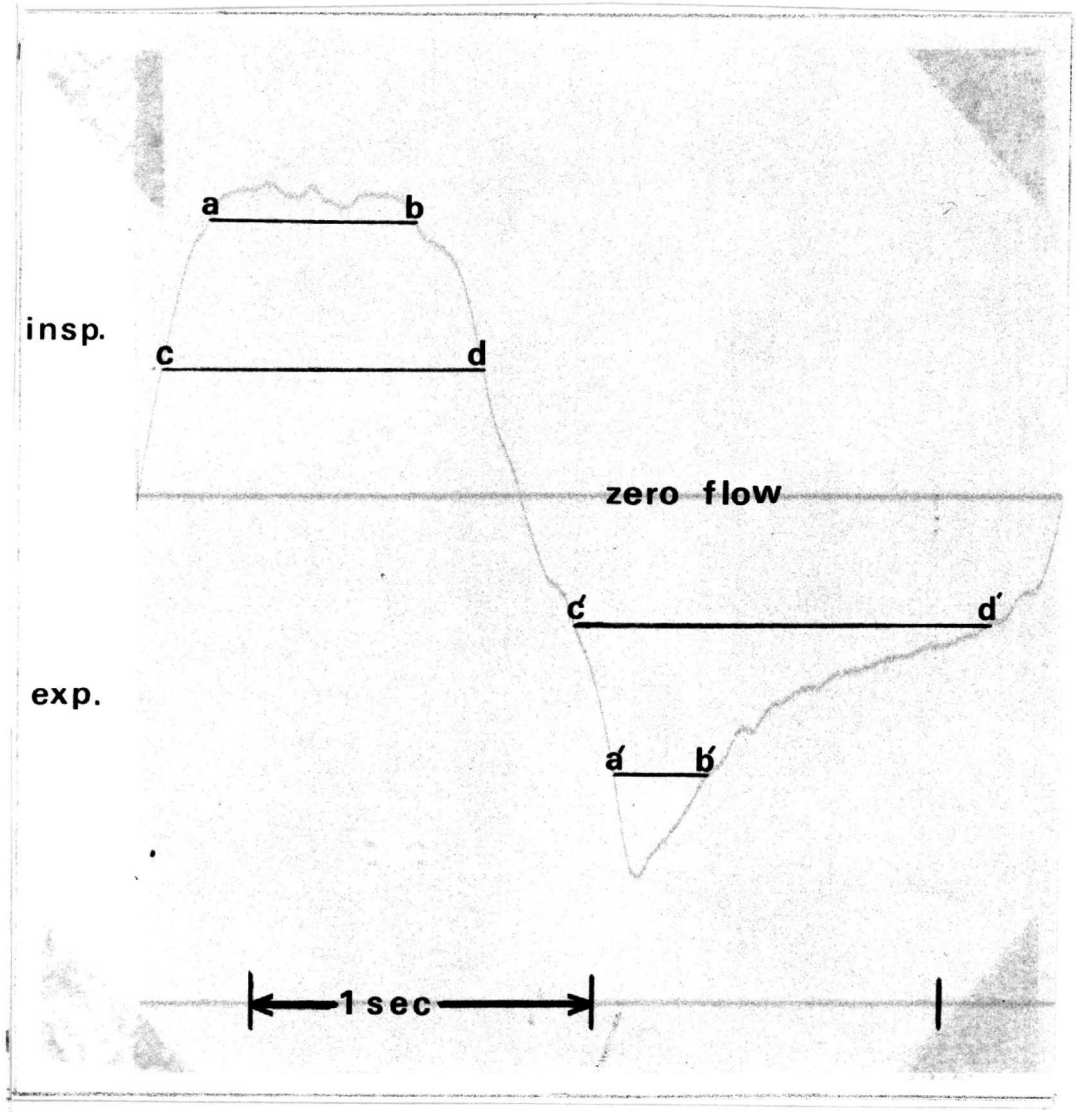


Fig 33

Duration of breath sounds related to the threshold of audibility. Breath sounds on the chest wall are audible at flow rates arbitrarily set by lines ab, a'b' Breath sounds at the mouth or trachea become audible at flow rates indicated by the line cd, c'd'.

acceleration phase followed by a period of relative steady flow and a deceleration phase. The expiratory flow curve shows only two phases: an acceleration and a slow deceleration phase without an intervening period of steady flow. The intensity of breath sounds is relatively constant in inspiration corresponding to the phase of steady flow. In expiration, on the other hand, the sound intensity is maximum in the early phase of expiration and falls off towards the end of the expiration (Fig. 32).

The breath sounds become audible whena certain minimum flow rate is reached during breathing. The turbulent eddies necessary to generate breath sounds are produced at a lower flow rate in the trachea than in the lobar and segmental bronchi which form the main source of breath sounds heard at the chest wall. In Fig. 33 the lines cd, c'd' arbitrarily represent the threshold flow rates above which breath sounds are heard over the trachea in inspiration and expiration. The lines ab, a'b' are the corresponding threshold flow rates for breath sounds heard on the lower regions of the chest wall in the two phases of respiration. It is seen that in inspiration because the flow rate is relatively steady, the duration for which breath sounds are heard at the trachea and on the lower regions of the chest wall are approximately equal (above lines cd, ab). In expiration, on the other hand, the duration of breath sounds on the lower regiond of the chest wall is restricted to early expiration (above line a'b') whereas the duration for which breath sounds are heard over the trachea occupy a large part of expiration (above line c'd').

Comparison of breath sounds recorded at the mouth and on the chest wall show that they are acoustically similar, that is, they are continuous sounds made up of random amplitudes. The difference in frequency distribution is due to selective filtration of higher frequencies during their transmission through the lung and the chest wall (Buller and Dornhorst, 1956). Thus breath sounds at the mouth are relatively less filtered with a frequency range between 200 and 2000 Hz, breath sounds recorded on the

left upper chest show a frequency distribution between 200 - 800 Hz, and breath sounds recorded on the posterior chest wall above the diaphragm show a narrow range of frequencies between 200 and 400 Hz.

SUMMARY

Breath sounds are continous sounds of no definite pitch. They contain a wide range of oscillations of random amplitude and frequency with approximately even distribution of energy. By analogy with white light, they are referred to as a respiratory white noise.

Breath sounds are generated by oscillations of the gas medium. They are produced in those airways where the gas flow is non-laminar. These include the upper airways, trachea and the proximal bronchi. It is suggested that breath sounds produced in these airways are transmitted at the mouth with a minimum loss of intensity and frequency. The intensity of inspiratory breath sounds at the mouth therefore closely reflect the total sound energy generated at source. Breath sounds heard at the chest are modified during their transmission through the lung and the chest wall. As a result some sound energy is lost and the higher frequencies filtered out. The sound intensity at the chest wall reflects sound energy generated mainly in the regional lobar and segmental bronchi with a limited contribution from the upper airways, trachea and the main bronchi.

At similar flow rates, the intensity of inspiratory breath sounds at the mouth is greater in patients with chronic bronchitis and asthma than in normal subjects. The loud breath sounds in these patients, it is suggested, are produced in narrowed proximal bronchi where the linear velocity of gas flow is increased. In general the louder the inspiratory breath sounds the lower the FEV_1. However, in primary emphysema, the inspiratory sound intensity at the mouth is normal in the face of a low FEV_1. It is suggested that the inspiratory calibre of the proximal intra-

thoracic airways where breath sounds are produced is normal in this disease. In tracheal stenosis, the inspiratory breath sounds are disproportionately loud while the FEV_1 is normal or slightly reduced. Hence measurements of inspiratory sound intensities and their comparisons with the results of tests of expiratory obstruction can help in the diagnosis of these conditions.

Bronchodilator drugs reduce inspiratory sound intensity when this is increased. Exercise and inhalation of tobacco smoke increase inspiratory sound intensity in some asthmatic subjects. The magnitude of reduction in sound intensity after bronchodilator drugs is of the same order as the increase in the FEV_1 and PEF.

At the bedside loudness of breath sounds at the mouth is best appreciated by listening with the unaided ear. The breathing of a normal subject is barely audible. The breath sounds of patients with chronic bronchitis and asthma are frequently loud. If the forced expiratory time is increased while the inspiratory sound intensity is normal, pure emphysema is likely. On the other hand, if the forced expiratory time is normal or slightly reduced while breath sounds are loud, this indicates stenosis of the upper airways, trachea or the main bronchus. The intensity of breath sounds as heard at the chest wall reflect the rate of air entry in the regional lobar and segmental bronchi and so to regional ventilation as well as the transmitting properties of the lung and the chest wall.

T A B L E - I

MICROPHONE OUTPUT (μV) AT 60 l/min INSPIRATORY FLOW RATE IN 12 NORMAL SUBJECTS

FIGURES REPRESENT MEAN OF 6 OBSERVATIONS

	1	2	3	4	5	6	7	8	9	10	11	12	Mean of all Observations
Microphone output at 60 l/min (μV)	45	54	41	53	59	52	58	64	59	44	39	61	52.4
S.D.	9.6	7.6	6.9	10.0	12.3	7.7	8.8	15.4	15.8	9.3	13.8	9.0	8.2

T A B L E - II

NORMAL SUBJECTS. RISE OF MICROPHONE OUTPUT (p) IN μV per 10 l/min INSPIRATORY FLOW RATE

FIGURES REPRESENT MEAN OF 6 OBSERVATIONS

	SUBJECT NO.												
	1	2	3	4	5	6	7	8	9	10	11	12	Mean of all Observations
p	5.8	10.0	13.0	7.2	9.8	8.7	14.1	10.7	9.7	14.5	5.1	10.8	10.1
S.D.	1.8	2.2	6.7	0.8	3.5	4.5	5.5	3.8	3.7	2.4	1.9	2.2	4.5

T A B L E - III

OBSERVER ERROR IN MEASUREMENT OF INSPIRATORY SOUND INTENSITY. FIGURES REPRESENT MEAN DIFFERENCE IN SOUND INTENSITY BETWEEN OBSERVER A, B AND C. POSITIVE SIGN INDICATE THAT OBSERVER A OVERESTIMATED SOUND INTENSITY COMPARED TO OBSERVER B AND C AND OBSERVER B OVERESTIMATED SOUND INTENSITY COMPARED TO OBSERVER C

	Observer A and B	Observer A and C	Observer B and C
No. of Observations	50	50	50
Mean Difference in Sound Intensity between two observers (db)	+ 0.7	+ 0.9	+ 0.24
S.D.	1.84	2.00	1.33

T A B L E - IV

COEFFICIENT OF CORRELATION (r) AND SIGNIFICANCE OF CORRELATION (P) OF INSPIRATORY SOUND INTENSITY WITH PEAK EXPIRATORY FLOW RATE (PEF), FORCED EXPIRATORY VOLUME IN ONE SECOND (FEV_1), VITAL CAPACITY (VC), AIRWAY RESISTANCE (R_{aw}) AND MAXIMUM MID-INSPIRATORY FLOW RATE (MMIF).

	PEF		FEV_1		VC		R_{aw}		MMIF	
	r	P	r	P	r	P	r	P	r	P
Chronic Bronchitis	- 0.60	< 0.001	- 0.60	< 0.001	- 0.63	< 0.001	+ 0.50	< 0.01	- 0.61	< 0.001
A s t h m a	- 0.56	< 0.001	- 0.63	< 0.001	- 0.44	< 0. 01	+ 0.47	< 0.05	- 0.53	< 0.01

T A B L E - V

COEFFICIENT OF CORRELATION (r) AND SIGNIFICANCE OF CORRELATION (P) OF INTENSITY OF INSPIRATORY BREATHS SOUNDS WITH AIRWAY RESISTANCE (R_{aw}), LOGARITHM OF AIRWAY RESISTANCE ($\log R_{aw}$), SPECIFIC AIRWAY RESISTANCE (SR_{aw}), AND LOGARITHM OF SPECIFIC AIRWAY RESISTANCE ($\log SR_{aw}$).

		R_{aw}	$\log R_{aw}$	SR_{aw}	$\log SR_{aw}$
Chronic Bronchitis	No of observations	33	33	33	33
	(r)	0.50	0.50	0.35	0.39
	(P)	< 0.01	< 0.01	> 0.05	< 0.05
Asthma	No of observations	18	18	18	18
	(r)	0.47	0.47	0.52	0.46
	(P)	> 0.05	> 0.05	< 0.05	> 0.05

T A B L E - VI

PHYSIOLOGICAL DATA ON 7 PATIENTS WITH PRIMARY EMPHYSEMA. PEF (PEAK EXPIRATORY FLOW RATE), FEV_1 (FORCED EXPIRATORY VOLUME IN ONE SECOND), R_{aw} (AIRWAY RESISTANCE) T_{co} (TRANSFER FOR CARBON MONOXIDE), TLC (TOTAL LUNG CAPACITY), I_s (INSPIRATORY SOUND INTENSITY). FIGURES IN BRACKETS REPRESENT PERCENTAGE OF THE PREDICTED NORMAL VALUE-

Case No.	Age (Yrs)	Sex	PEF (l/min)	FEV_1 (litres)	R_{aw} (cmH2o/l/sec)	T_{co} (ml/min/mm/Hg)	TLC (litres)	I_s (db)
1	65	M	125 (25)	0.8 (30)	2.5	6 (24)	6.5 (24)	0
2	53	M	155 (28)	0.8 (24)	2.0	10 (43)	9.1 (136)	0
3	55	M	180 (32)	2.0 (57)	1.5	15 (49)	8.9 (122)	2
4	40	M	100 (16)	0.7 (20)	-	7 (22)	8.5 (130)	0
5	56	M	300 (53)	2.3 (66)	1.4	16 (31)	8.5 (113)	0
6	59	M	120 (22)	0.8 (27)	1.6	10 (27)	7.6 (117)	0
7	54	F	180 (31)	1.2 (43)	3.7	8 (24)	6.1 (109)	6

T A B L E - VII

BRONCHIAL AND TRACHEAL STENOSIS. PEF AND FEV_1 COMPARED WITH INSPIRATORY SOUND INTENSITY

FIGURES IN BRACKETS REPRESENT PREDICTED VALUES

Code No.	Age (Yrs)	Sex	Diagnosis	PEF (litres/min)	FEV_1 (litres)	Sound Intensity (db)
1	54	M	Stenosis left lower lobe bronchus	440 (440)	2.8 (2.9)	12
2	75	M	Left upper lobe bronchial stenosis	260 (550)	2.1 (3.1)	17
3	45	F	Stenosis left main bronchus	290 (410)	1.9 (2.6)	19
4	24	F	Bronchial and laryngeal stenosis	180 (430)	2.3 (2.8)	28
5	45	F	Right upper lobe bronchial stenosis. Chronic Bronchitis	140 (420)	1.2 (2.6)	23
6	22	F	Tracheal stenosis	200 (450)	2.4 (2.9)	32

T A B L E - VIII

TRACHEAL STENOSIS. CHANGE IN INSPIRATORY SOUND INTENSITY BEFORE AND AFTER TRACHEAL DILATATION COMPARED WITH CHANGE IN THE RATIO FEV_1 % OF PREDICTED VALUE, /PEF % OF PREDICTED VALUE R_{aw} (AIRWAY RESISTANCE), SR_{aw} (SPECIFIC AIRWAY RESISTANCE), AND MMIF (MAXIMUM MID-INSPIRATORY FLOW RATE).

	$\frac{FEV_1\ \%\ Pre\ Value}{PEF\ \%\ Pre\ Value}$	R_{aw} (cm H_2o/l/sec)	SR_{aw} (cm H_2o sec)	MMIF (l/min)	Inspiratory sound Intensity (db)
Before tracheal dilatation	1.87	3.5	9.8	200	32
2-weeks after tracheal dilatation	1.89	1.7	6.1	250	20
4-weeks after tracheal dilatation	1.67	1.4	4.6	300	15

T A B L E - IX

CHANGE IN PEF, FEV_1 AIRWAY RESISTANCE (R_{aw}), MAXIMUM MID-INSPIRATORY FLOW RATE (MMIF) AND SOUND AMPLITUDE FOLLOWING INHALATION OF ISOPRENALINE AEROSOL, EXPRESSED AS A PERCENTAGE OF THE INITIAL VALUE

		PEF	FEV_1	R_{aw}	MMIF	SOUND AMPLITUDE
Chronic Bronchitis	No. of Observations	34	36	24	30	36
	Mean	26%	15%	21%	28%	27%
	Range	2- 81	0 - 70	0 - 48	0 - 126	0 - 92
	S.D.	17.9	13.5	15.4	32.3	24.7
A s t h m a	No. of Observations	31	31	12	26	31
	Mean	36%	23%	27%	22%	23%
	Range	0 - 132	0 - 169	0 - 42	0 - 71	0 - 76
	S.D	28.3	36.1	11.3	23.2	19.5

T A B L E - X

CHNAGE IN PEF, FEV_1 AND SOUND AMPLITUDE IN PATIENTS WITH CHRONIC BRONCHITIS FOLLOWING ADMINISTRATION ATROPINE. FIGURES REPRESENT PERCENTAGE CHANGE OF INITIAL VALUE

	PEF	FEV_1	Sound Amplitude
No. of observations	18	18	18
Mean Change (%)	33	26	36
Range (%)	0 - 75	0 - 89	0 - 89
S.D.	23.0	20.7	24.9

T A B L E - XI

ATROPINE RESPONSE IN CHRONIC BRONCHITIS. FIGURES REPRESENT CHANGE AFTER ATROPINE AS PER CENT OF TOTAL CHANGE AFTER ATROPINE AND ISOPRENALINE

Case No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	Mean	S.D.
FEV_1 (%)	100	61	60	86	67	100	0	100	100	75	100	100	100	83	67	75	79.6	26.3
Sound Amplitude (%)	100	57	95	21	44	100	100	100	71	70	100	100	100	100	0	78	77.3	31.3

TABLE - XII

CHANGE IN PEF, FEV_1 AIRWAY RESISTANCE (R_{aw}) AND SOUND INTENSITY AFTER ATROPINE AND ISOPRENALINE IN ONE SUBJECT WITH DIFFUSE AIRWAY OBSTRUCTION

	PEF (l/min)	FEV_1 (litres)	R_{aw} (cm H_2o/l/sec)	Sound Intensity (db)
Resting	160	1.3	2.4	15
After Atropine	250	1.5	1.5	15
After Isoprenaline	230	1.7	1.6	15

T A B L E - XIII

CHANGE IN FEV_1 AND INSPIRATORY SOUND INTENSITY AFTER EXERCISE PROVOCATION AND SUBSEQUENT ADMINISTRATION OF ISOPRENALINE AEROSOL IN PATIENTS WITH EXERCISE INDUCED ASTHMA

Case No.	Age (Yrs)	Sex	FEV_1 (litres)			Inspiratory Sound Intensity (db)		
			Before Exercise	After Exercise	Isoprenaline	Before Exercise	After Exercise	Isoprenaline
1	21	F	1.9	1.1	2.6	4	20	3
2	23	M	3.2	2.2	3.6	5	16	5
3	16	M	5.0	3.4	4.6	0	13	2
4	16	M	3.8	2.1	4.2	3	15	10
5	44	M	3.5	3.2	3.4	8	14	11
6	51	M	1.7	1.8	1.9	11	17	12

T A B L E - XIV

EFFECT OF ATROPINE AND ISOPRENALINE ON FEV_1 AND INSPIRATORY SOUND INTENSITY IN A PATIENT WITH EXERCISE INDUCED ASTHMA

	Before Exercise	After Exercise	Before Atropine	Exercise 1-hour After Atropine	Before Isoprenaline	Exericse 5-minutes After Isoprenaline
FEV_1	3.2	2.2	3.3	2.3	3.1	3.6
Sound Intensity (db)	5	16	5	17	5	5

T A B L E - XV

PEF, FEV_1 , INSPIRATORY SOUND INTENSITY AND DIAMETER OF STOMA OF LARYNGECTOMY PATIENTS. FIGURES IN BRACKETS REPRESENT PREDICTED VALUES.

Case No.	Age (Yrs)	Sex	Size of Stoma		PEF (l/min)	FEV_1 (litres)	Sound Intensity (db)
			Vertical Diameter (cm)	Horizontal Diameter (cm)			
1	63	M	2.0	2.0	260 (490)	1.8 (2.6)	18
2	66	M	1.0	1.0	230 (500)	1.3 (2.8)	16
3	70	M	1.0	1.5	280 (490)	1.5 (2.6)	15
4	68	M	-	-	-	-	22
5	53	M	2.0	0.5	-	-	13
6	53	M	2.0	0.5	-	-	10

T A B L E - XVI

COMPARISON BETWEEN PEF, FEV_1, VITAL CAPACITY (VC) AND SOUND INTENSITY IN 3 SUBJECTS FOLLOWING PNEUMOECTOMY. FIGURES IN BRACKETS REPRESENT PREDICTED VALUES

Case No.	Age	Sex	PEF (l/min)	FEV_1 (litres)	VC (litres)	Sound Intensity (db)
1	52	F	145 (380)	1.2 (2.6)	2.1 (2.9)	18
2	60	F	110 (350)	0.7 (2.0)	1.0 (2.5)	30
3	64	M	180 (530)	1.2 (2.9)	2.0 (4.0)	28

T A B L E - XVII

INSPIRATORY SOUND INTENSITY BREATHING AIR AND HELIUM

(ONE PATIENT SHOWING NO CHANGE IN SOUND INTENSITY IS OMITTED)

Case No.	Diagnosis	INSPIRATORY SOUND INTENSITY (db)		Mean change in Inspiratory Sound Intensity (db)	Range (db)
		Air	Helium		
1	Asthma	20	16	9	4 - 14
2	Asthma	15	4		
3	Chronic Bronchitis	27	20		
4	Laryngectomy	13	0		
5	Laryngectomy	15	10		
6	Pneumonectomy	30	16		

T A B L E - XVIII

EFFECT OF SMOKING A CIGARETTE ON FEV_1 AND INSPIRATORY SOUND INTENSITY (I_s) IN NORMAL AND ASTHMATIC SUBJECTS

	Case No.	Before Smoking		After Smoking	
		FEV_1 (litres)	I_s (db)	FEV_1 (litres)	I_s (db)
Normal	1	2.5	0	2.5	0
	2	5.2	5	5.2	8
	3	4.8	0	4.9	0
Asthma	4	1.3	11	0.9	16
	5	2.1	16	2.2	18
	6	2.9	16	2.7	16
	7	0.9	16	0.9	15
	8	-	4	-	14

REFERENCES

Altounyan, R.E.C. (1964) Variations of drug action on airway obstruction in man. Thorax, 19, 406

Andral, C. (1836) The Clinique Medicale. Translated by D. Spillen, p. 290, Henry Renshaw, London.

Beau, J.H.S. (1834) Recherches sur la cause bruits respiratoires percus au moyen de l'auscultation Arch. gen. Med., 5, 557.

Black, L.F., Hyatt, R.E., and Stubbs, S.E. (1972) Mechanism of expiratory airflow limitation in chronic obstructive pulmonary disease associated with alpha 1 - antitrypsin deficiency. Amer. Rev. Dis. 105, 891.

Bentivoglio, L.C., Beerel, F., Bryan, A.C., Stewart, P.B., Rose, B., & Bates, D.V. (1963) Regional pulmonary function studied with Xenon 123 in patients with bronchial asthma. J. Clin. Invest., 42, 1193.

Bondet, A., & Chauveau, A (1887) Bruits respiratoires normaux et anormaux. Arch. gen. Med., 1, 11.

British and Foreign Medico-Chirurgical Review. (1873) On the physical theory of murmurs; vascular, cardiac respiratory. British and Foreign Medico-Chirurgical Review, 52, 15.

Bullar, J.P. (1884) Experiments to determine the origin of the respiratory sounds. Proc. Roy. Soc., 37, 411.

Buller, A.J. & Dornhorst, A.C. (1956) The Physics of some pulmonary signs. Lancet, 2, 649.

Bushnell, G.E. (1921) The mode of production of the so-called vesicular murmurs of respiration. J. Amer. Med. Ass; 77, 2104.

Cabot, R.C., & Dodge, H.F. (1925) Frequency characteristics of heart and lung sounds. J. Amer. Med. Ass; 84, 1793.

Caelius Aurelianus. (1950) On acute Diseases and on Chronic Diseases. Edited and Translated by I.E. Drabkin, Acute Diseases, Book 2, p.711, University of Chicago Press, Chicago.

Dubois, A.B., Botelho, S.Y., & Comroe, J.H. Jnr. (1956) A new method for measuring airway resistance in man using a body plethysmograph. J. Clin. Invest., 35, 327.

Fahr, C. (1927) The accousticsof the bronchial breath sounds. Arch. intern. Med., 39, 286.

Flint, A. (1856) Physical Exploration and Diagnosis of Disease Affecting the Respiratory Organs, p.175, Blanchard and Lea, Philadelphia.

Forgacs, P. (1967) Crackles and Wheezes. Lancet 2, 209.

Forgacs, P. (1969) Lung Sounds Brit. J. Dis. Chest, 63, 1.

Forgacs, P., Nathoo, A.R., & Richardson, H.D. (1971) Breath sounds. Thorax, 26, 288.

Gee, S. (1877) Auscultation and Percussion Together with the other Methods of Physical Examination of the Chest, 2nd edition, p. 129, Oxford University Press, London.

Guyatt, A.R., Berry, C., Alpers, J.H., Bramley, A.C. & Fletcher, C.M. (1970) Relationship of airway conductance and its immediate change on smoking to smoking habits and symptoms of chronic bronchitis. Amer. Rev. Resp. Dis., 101, 44.

Hooke, E. (1705) The Posthumous Works of Dr. Robert Hooke Containing his Cutlerian Lectures and other Discourses, p.39. Richard Waller, London.

Jaeger, M.J. & Matthys, H. (1970). The pressure flow characteristics of the human airways. Airway Dynamics, p.21. Charles C. Thomas, Springfield, Illinois.

Laennec, R.T.H. (1819). Translation of selected Passages from De l'Auscultation Mediate (First Edition). With a biography by Sir William Hale-White, No. 173, p.75, John Bale, Sons & Danielson, London, 1923.

Laennec, R.T.H. (1819). Translation of Selected Passages from De l'Auscultation Mediate (First Edition). With a biography by Sir William Hale-White, No. 183, p.79, John Bale, Sons & Danielson, London, 1923.

Leblanc, P., Maclem, P.T. & Ross, W.R.D. (1970). Breath sounds and distribution of pulmonary ventilation. Amer. Rev. Resp. Dis., 102, 10.

Martini, P. (1922). Studien uber Perkussion and Auskultation. Dtchr. Arch. klin. Med., 139, 257.

Martini, P., Muller, H. (1923). Studien uber das Bronchialatmen. Dtchr. Arch. klin. Med., 143, 159.

McKusick, V.A., Jenkins, J.T., & Webb, G.N. (1955). The accoustic basis of the chest examination. Amer. Rev. Tuberc. 72, 12.

Mead, J., Turner, J.M., Maclem, P.T., & Little, J.B. (1967). Significance of the relationship between lung recoil and maximal expiratory flow. J. Appl. Physiol. 22, 95.

Merat, F.V. (1819). Pectoriloque. In Dictionnaire des Sciences Medicales par une Societe de Medecins et de Chirurgiens, 40, 9-35 Paris.

Mills, R.J. (1969). The Mechanical effect of the heart beat on the plethysmographic pressure. Progr. Resp. Res., 4, 164.

Montgomery, C.M.L. (1938). The transmission of sounds through the chest. In Diseases of the Chest and the principles of physical Diagnosis, by G.W. Norris and H.R.M. Landis, 6th edition, p.63, W.B. Saunders, Philadelphia.

Nairn, J.R. & Turner-Warwick, M. (1969). Breath sounds in emphysema. Brit. J. Dis. Chest., 63, 29.

Norris, G.W. & Landis, G.R.M. (1940). Diseases of the Chest and the Principles of Physical Diagnosis, 6th edition, revised, p. 121, W.B. Saunders, Philadelphia.

Palmiere, C.C. (1953). Ulteriori raffronti radiochimografici ed elettromanometrici sulla meccancia dei diversi territori polmonari nella piccola respiraxione cardiaca. Minerva med., 44, 1655.

Palmieri, C.C., Petrucci, D., Lura, A. (1953) Ricerche comparative radiochimografiche ed elettromanometriche sulla cinematica e sulla dinamica dei diversi territori polmonari. Minerva. med., 44, 125.

Pedley, T.J., Schroter, R.C., & Sudlow, M.F. (1970) The prediction of pressure drop and variation of resistance within the human bronchial airways. Resp. Physiol., 9, 387.

Pratt, J., & Bushnell, G.E. (1925) Physical Diagnosis of Diseases of the Chest, p.36, W.B. Saunders, Philadelphia.

Pride, N.B. (1971) The assessment of airflow obstruction. Brit. J. Dis. Chest, 65, 135.

Ramadge, F.H. (1847) Asthma, its variations and complications: Or, Researches into the Pathology of Disordered Respiration; etc, 2nd edition p.42, Longman, Brown Green and Longmans, London.

Sahli, H. (1892)Ueber die Entstehung des vesciularathmens. Cor. Bl. Schweiz. Aerzte, 22, 265.

Skoda, J. (1853) A treatise on Auscultation and Percussion. Translated from the 4th edition by W.O. Markham, p.102, Highley & Son, London.

Spittal, R. (1839) Experiments and observations on the cause of the sounds of respiration. Edinb. med. surg. J., 51, 99.

Sterling, G.M. (1967) Mechanism of bronchoconstriction caused by cigarette smoking. Brit. med. J., 3, 275

Stokes, W. (1837) A treatise on the Diagnosis and Treatment of Diseases of the Chest. Part 1: Diseases of the Lung and Widpipe, p.251, Hodges and Smith, Dublin.

Walshe, W.H. (1871) A Practical Treatise on the Diseases of the Lungs: Including the principles of Physical Diagnosis, etc, 4th edition, p.90, James Walton, London.

West, J.B., & Hugh-Jones, P. (1961) Pulsatile gas flow in bronchi caused by the heart beat. J. Appl. Physiol. 16, 697.

Williams, C.J.B. (1840) Patholgy and Diagnosis of Diseases of the Chest etc, 4th edition, p.22, John Churchill, London.

ACKNOWLEDGEMENTS

I wish to thank

Dr. T.J.H. Clark for his help and encouragement.

Dr. L.H. Capel for his interest and valuable discussions.

Mr. P.J. Bishop for valuable assistance with the references.

Mr. D. Richardson for guidance and technical assistance with the recording of breath sounds.

Miss P.T. D'Cruze and Mrs. E.D. Bird for their invaluable help with the manuscript.

Mr. K. Mormon, Miss L. Pegus and Mrs. A. Cole for their help with illustrations.