SPIROMETRIC EVALUATION OF OCCUPATIONAL RESPIRATORY DYSFUNCTION AMONG HOSPITAL SANITARY WORKERS IN A TERTIARY CARE CENTRE

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M.D. (PHYSIOLOGY)

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CHENGALPATTU MEDICAL COLLEGE,

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILNADU

MAY 2020

CERTIFICATE

This is to certify that this dissertation titled "**SPIROMETRIC EVALUATION OF OCCUPATIONAL RESPIRATORY DYSFUNCTION AMONG HOSPITAL SANITARY WORKERS IN A TERTIARY CARE CENTRE** '' is a bonafide record work done by **Dr.S.EZHILARASI**, during the period of her postgraduate study from May 2017 to May 2020 under guidance and supervision in the Department of Physiology, Chengalpattu Medical College and Hospital, Chengalpattu – 603 001 in partial fulfilment of the requirement for **M.D. PHYSIOLOGY** degree Examination of The Tamil Nadu Dr. M.G.R. Medical University.

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I declare that the dissertation entitled "**SPIROMETRIC EVALUATION OF OCCUPATIONAL RESPIRATORY DYSFUNCTION AMONG HOSPITAL SANITARY WORKERS IN A TERTIARY CARE CENTRE**" submitted by me for the degree of M.D. is the record work carried out by me during the period of **May 2017 to May 2020** under the guidance of **Dr.A.ANITHA, M.D., DCH.,** Professor Vice Principal, Chengalpattu Medical College, Chengalpattu. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the University regulations for the award of degree of M.D., Physiology (Branch V) examinations to be held in May 2020.

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LIST OF ABBREVIATIONS

HCW	Health care waste
BMW	Bio medical waste
GHG	Greenhouse gas
CPCB	Central pollution control board
HSW	Hospital sanitation worker
PPE	Personal protective equipment
MALT & BALT	Mucosa & Bronchi associated lymphoid tissue
FVC	Forced vital capacity
FEF	Forced expiratory capacity
FET	Forced expiratory time
FRC	Functional residual capacity
TLC	Total lung capacity
ASSOCHAM	Associated Chambers Of Commerce And Industry Of India
COPD	Chronic obstructive pulmonary disease
CFU	Colony forming unit
AM	Alveolar macrophages
ATS	American Thoracic Society

INSTITUTIONAL ETHICAL COMMITTEE

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SPIROMETRIC EVALUATION OF OCCUPATIONAL RESPIRATORY DYSFUNCTION AMONG HOSPITAL SANITARY WORKERS IN ATERTIARY CARE CENTRE.

AUTHORS: Dr.S.Ezhilarasi[1], Dr.A.Anitha[2]

INSTITUTION: Department of Physiology, Chengalpattu Medical College

BACKGROUND: Working in dusty environment face the risk of inhaling particulate materials that may lead to adverse respiratory effects. Sanitary workers are exposed to a number of pathogens, toxic substances, chemicals that come from the waste itself and from its decomposition. As a result of their exposure to multiple risk factors, they suffer high rates of occupational health problems, which would definitely alter the pulmonary functions and respiratory endurances. Individuals who breathe through their mouth have higher pulmonary ventilation rates when comparing to those who breathe through their nose. This is likely to be attributed to the occupational exposure of this group to workplace contaminants, particularly bio aerosols.

So this study was done to evaluate the respiratory dysfunction among sanitary workers who are exposed to environmental and occupational hazards**.**

AIM & OBJECTIVE: To evaluate the occupational respiratory dysfunction among sanitary workers.

1. To evaluate the respiratory functions in sanitary workers and normal healthy individuals.

2. To compare the effect of duration of exposure on respiratory functions in sanitary workers.

MATERIALS & METHODS: A cross sectional study was done after IEC approval, with written informed consent on 120 individuals of age group 20-45 years. Group I- 60 sanitary workers involved in waste collection and disposal of both gender. GROUP II- 60 healthy non exposed candidates, age and gender matched. Detailed history and clinical examination was carried out to rule out any acute or chronic illness. Information regarding respiratory illness, frequency and symptoms noted. The pulmonary function parameters; Forced Vital Capacity [FVC], Forced Expiratory Volume in 1 second [FEV1] ,Forced Expiratory Flow [FEF25–75] Peak Expiratory Flow [PEF] were recorded using spirometer ,according to the American Thoracic Society criteria. Parameters were compared using SPSS16.0 version.

RESULTS: All pulmonary function parameters were reduced in sanitary workers compared to control group. FVC%, FEV1 was reduced significantly.

CONCLUSION: The lung functions are commonly affected due to occupational exposure in sanitary workers.

Key words: sanitary workers, pulmonary function, FVC%, FEV1.

INTRODUCTION

The birth of civilisation has led to developments in all walks of human life to meet various human needs. The scientific discoveries have made great achievements in quality of health care delivery systems. But less concentrated, other side of the coin is confronting us with huge demand to tackle the problem – **Health care waste (HCW) disposal.**

Population explosion and urbanisation has led to generation of huge quantities of solid wastes. Open dumping has been the common mode of waste disposal without segregation till now. Due to which environmental pollution poses worldwide threat to human health. Human activity aims at deriving benefits from raw materials but creating left over complexities[(1)]. Complexity in waste is also increasing with bio and non- biodegradable waste.

Health care waste

It shall mean discarded (and untreated) materials from health-care activities on humans or animals, which have the potential of transmitting infectious agents to humans[(2)].

The emergence of AIDS during the 1980s drew our attention towards blood borne diseases. Most people now cannot imagine handling blood without wearing appropriate gloves. The SARS epidemic and its aftermath in china in 2003 further showed our increased responsibility towards other infectious diseases, in work place health and safety programs. Concern is also raised when

the public is visually exposed to HCW, as in the case of healthcare waste found washed up along the north-eastern US shoreline in 1988. Similarly, public awareness has arisen in other areas of the world. In the state of West Bengal, India, the poor management of health-care waste caused several institutions to consider returning to reusable glass syringes rather than to continue with single-use plastic syringes. The reuse of unsterilized syringes has been estimated to cause 8-16 million cases of hepatitis B ,2.3 to 4.7 million cases of hepatitis C and 80,000 to 160,000 cases of Human Immunodeficiency viruses infections per year[(3)].

GLOBAL BURDEN

World Bank report has expressed alarm over the growing piles of municipal garbage across cities of the world and their disposal, which are a source of greenhouse gas (GHG) emissions. It says, the global municipal solid waste generation will increase by 70% from the current 1.3 billion tonnes per year to 2.2 billion tonnes in 2025. Of the 171 countries, 61, including India, have no data on municipal solid waste collection and disposal. The CPCB (Central Pollution Control Board) India, report shows only 10 out of 34-29% SPCBs (State Pollution Control Board and pollution control committees (PCCs) filed their reports in time by September 2009, and even by April 30, 2010, only 17 of them (50%) filed their reports . Even an otherwise progressive state like Tamil Nadu reported 99.64 per cent open-dumping in 841 out of its total 844 municipalities[(4)] .

Medical waste treatment typically involves four main goals

(1) Inactivate or destroy infectious pathogens or microbes;

(2) Destroy sharps;

(3) Render waste unrecognizable for ethical and confidentiality considerations; and

(4) Reduce the volume of waste

Other waste streams generated by hospitals, such as discarded PPE (Personal protective equipment's), excess prescription medication, chemical wastes, and radioactive materials may have adverse effects on both people and the environment, though,they generally do not pose risk of infection[(5)] .

To ensure safe and proper disposal ,the **Biomedical waste management rules 2016**, and KAYAKALP 2015[(6)] published by the Central Pollution Control Board (CPCB), Government of India in accordance with the spirit of the Environment (Protection) Act, 1986 provides the regulatory frame work for management of bio-medical waste generated in India though BMW comprises around 1% of total waste generated as it needs special handling.

The act defines "**Biomedical waste" (BMW)** as any waste, which is generated during the diagnosis, treatment or immunization of human beings or animals or research activities pertaining thereto or in the production or testing of biological or in health camps. The act further classifies biomedical waste into 10

major categories and lays down a system of colour coding for the purposes of segregation, handling, transportation and disposal.

The act makes it mandatory for the "occupier" (a person having administrative control over the institution and the premises generating bio-medical waste) to ensure strict adherence to the established standards while “handling” (includes the generation, sorting, segregation, collection, use, storage, packaging, loading, transportation, unloading, processing, treatment,destruction, conversion, or offering for sale, transfer, disposal) the generated waste[7,8].

KAYA KALP 2015 (Govt of India) guidelines focus on strengthening and streamlining of proper selection and maintenance of infrastructure, development of suitable policies for housekeeping services, selection & training of manpower, development and implementation of suitable cleaning methods in the form of protocols / SOP’s, effective supervision and monitoring by adequate staff and in-built mechanisms in the contracts coupled with an organizational structure which puts a premium on good housekeeping and sanitation. They also describe the structure of the housekeeping department / service, roles & responsibilities of workers &supervisors, qualification, experience & training needs of sanitation staff, equipment details for mechanized cleaning, chemicals & cleaning agents to be used, etc[9].

“**Housekeeping** is a support service department in a hospital, which is responsible for cleanliness, maintenance & aesthetic upkeep of patient care areas,

public areas and staff areas"[9]. The house keeping workers are engaged in medical waste disposal along with general waste generated in hospitals.

Table 1: Hazards covered in health sectors [6]

Section 1	Biological hazards	Blood-borne pathogens include viruses and bacteria which cause hepatitis B and C, HIV, latex, medical waste management, methicillin-resistant staphylococcus aureus,tuberculosis, and other airborne pathogens.
Section 2	Chemical hazards	Cleaning agents, ethylene oxide, formaldehyde, glutaraldehyde, mercury, methyl methacrylate, surgical smoke
Section 3	Ergonomic hazards	Computer workstations, hand-held devices, laboratory,laparoscopy,radiology,safepatienthandling,slips,trips,andfalls, sonography
Section 4	Hazardous drugs	Aerosolized medication, anaesthetic gases, antineoplastic drugs, nitric oxide, pentamidine, ribavirin
Section 5	Radiation	Ionizing radiation (radionuclides in nuclear medicine and diagnostic imaging, radionuclides in radiation therapy, X-rays), and nonionizing radiation (magnetic resonance imaging, lasers, ultraviolet lights)
Section 6	Psychological hazards	Shift work, stress, and violence

Hospital sanitation worker (HSW) is an important part of healthcare. Without them, we would not have the safe, clean environment we rely on in hospitals. Disease and infection would be rampant and patient care would be compromised[10]. But they belong to low socio economic group, needless to say underprivileged in all aspects. So their concentration lies in living and attitude

towards their job risks, and usage of personal protective equipment (PPE) are often neglected. Neither do they report any symptoms nor do they approach for treatment. Most of them are contract based, so drop outs out of sickness-absenteeism is overlooked.

Controlling and minimizing workplace hazards for healthcare personnel in hospitals present a unique challenge because the health and wellbeing of hospital patients must also be considered[6].

'**It's pretty dangerous to be a garbage man'** is quite true[11] . Products such as bleach, glass cleaner, detergents and air fresheners exacerbated asthma-related symptoms for the women, and their reduced lung function lasted until the morning after exposure, in some cases getting worse with time.

This shows the importance of developing workplace health and safety practices designed to limit exposures to irritant chemicals in cleaning products. Mixing cleaning products that contain bleach and ammonia can cause severe lung damage. In a big medical centre, the workers may be unionized and safety regulations will probably be more strongly adhered to, still, the type of protective gear will make a difference. Particle masks are not too expensive or cumbersome to use, but they don't keep the out fumes. To keep out the fumes, they may need more cumbersome equipment[12]. The occupational health hazards reported as respiratory followed by musculo-skeletal, dermatological, gastrointestinal,

injuries and nose and eye problems. The highest occupational health complaint was found in respiratory system[13].

THE RESPIRATORY SYSTEM

Lungs are the organs of gas exchange, providing oxygen to tissues and removing CO_2. It takes part in host defence acting as a primary barrier between environment and human body.

PHYSIOLOGIC STRUCTURE

The lungs are contained in a space with a volume of approximately 4 L. Airflow through respiratory system is by 3 interconnecting structures – upper airways, conducting airways and alveolar airway (also called as lung parenchyma) [14].

UPPER AIRWAY

It includes -Nose, sinuses, posterior pharynx, larynx up to vocal cords. Major function is to "condition" inspired air so that by the time it reaches the trachea, to body temperature and humidity. The nose filters, entrap, and clear particles larger than 10 microns in size.In humans, the volume of air entering the nares each day is on the order of 10,000 to 15,000 L. Nasal resistance increases with viral infections and with increased airflow, such as during exercise. When nasal resistance becomes too high, mouth breathing begins[14].

The interior of the nose is lined by respiratory epithelium interspersed with surface secretory cells which produce important immunoglobulins, inflammatory mediators, and interferons, which are the first line in host defence.

Sinuses are lined by ciliated epithelium, facilitating the flow of mucus from the upper airways and clear the main nasal passages approximately every 15 minutes. The ostia are readily obstructed in the presence of nasal edema, and retention of secretions and secondary infection (sinusitis) can result.

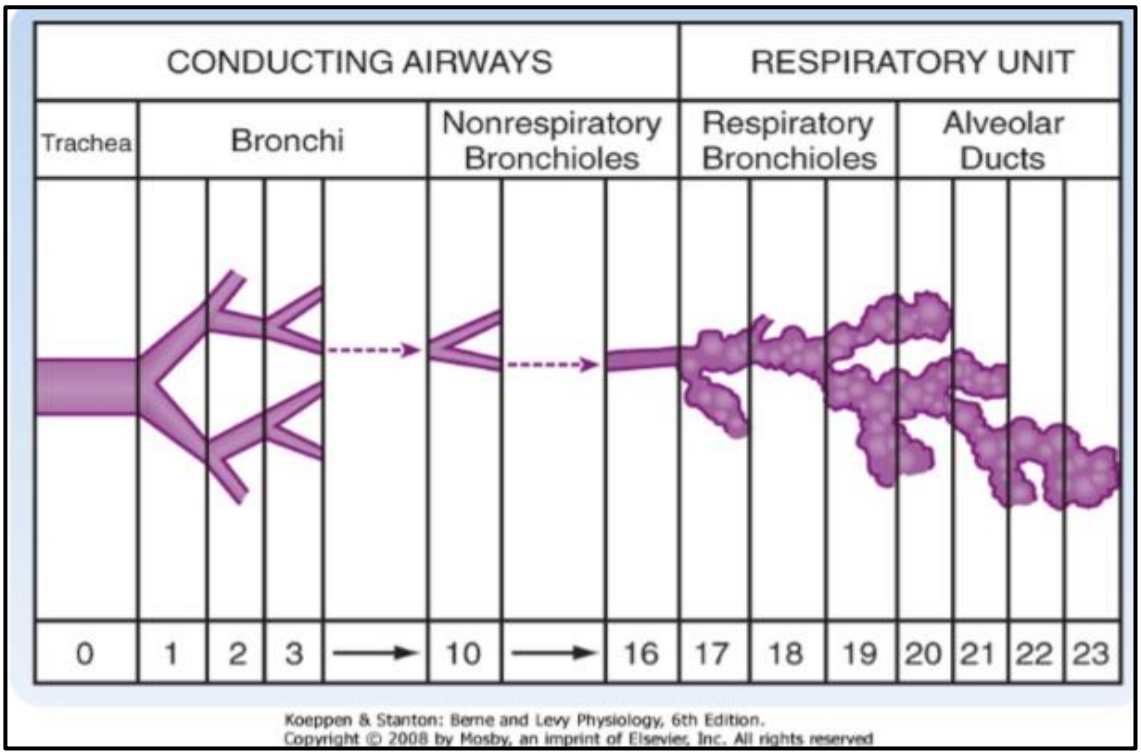


Figure 1 : Conducting airways and alveolar units of the lung

ALVEOLAR AIRWAY (LUNG PARENCHYMA)

This includes last 7 generations and made up of transitional respiratory bronchioles, alveolar duct and alveoli. There are around 300 million alveoli in

human lungs. The surfactants released by alveolar cells decreases the surface tension. Interstitium is a microscopic anatomical space bound by basement membrane of epithelial cell of alveoli and endothelial cell of pulmonary capillaries. It consists of collagen and reticulin fibers which create a helical network of connective tissue around the alveoli and respiratory airway walls. Both the lungs and chest wall are elastic in nature and they can expand and recoil. This elasticity is conferred by elastic tissue in airway and alveolar wall, and also by connective tissue in the inter alveolar space and by surfactant.

RESPIRATORY UNIT

The lung demonstrates anatomic and physiological unity. The layers of the structure through which exchange of gases takes place are

1) Fluid layer containing surfactant
2) Layer of alveolar epithelium
3) Epithelial basement membrane
4) Thin interstitial space between alveoli and capillaries
5) Capillary basement membrane
6) Endothelial cell layer of capillaries

PULMONARY DEFENSE MECHANISM

At all levels of respiratory tract specific and nonspecific defence mechanisms exists to protect the respiratory system.

1. Epithelial cells of conducting airway secrete IgA, surfactant protein A&B, various proteases, peptidases that kills the microbes directly. They also secrete chemokines and cytokines that attract immune cells there by killing the microbes indirectly.

2. The dichotomous branching of airway traps the smaller particles and clears it by coughing and mucociliary escalation.

3. The alveoli have pulmonary alveolar macrophages which secrete cytokines to attract the granulocyte and initiate immunological reaction. But this action is a two edged sword for the pulmonary alveolar macrophages may also release lysosomal products in extracellular space to cause inflammation of the interstitium that heals with fibrosis[15,16].

4. MUCOCILIARY CLEARANCE SYSTEM- protects the lower respiratory system by trapping and removing inhaled pathogenic viruses and bacteria, in addition to nontoxic and toxic particulates (e.g., pollen, ash, mineral dust, mold spores, and organic particles), from the lungs

The three major components of the mucociliary clearance system are two fluid layers referred to as the sol (periciliary fluid) and gel (mucus layer) phases and the cilia, which are positioned on the surface of the airway epithelial cells(14)

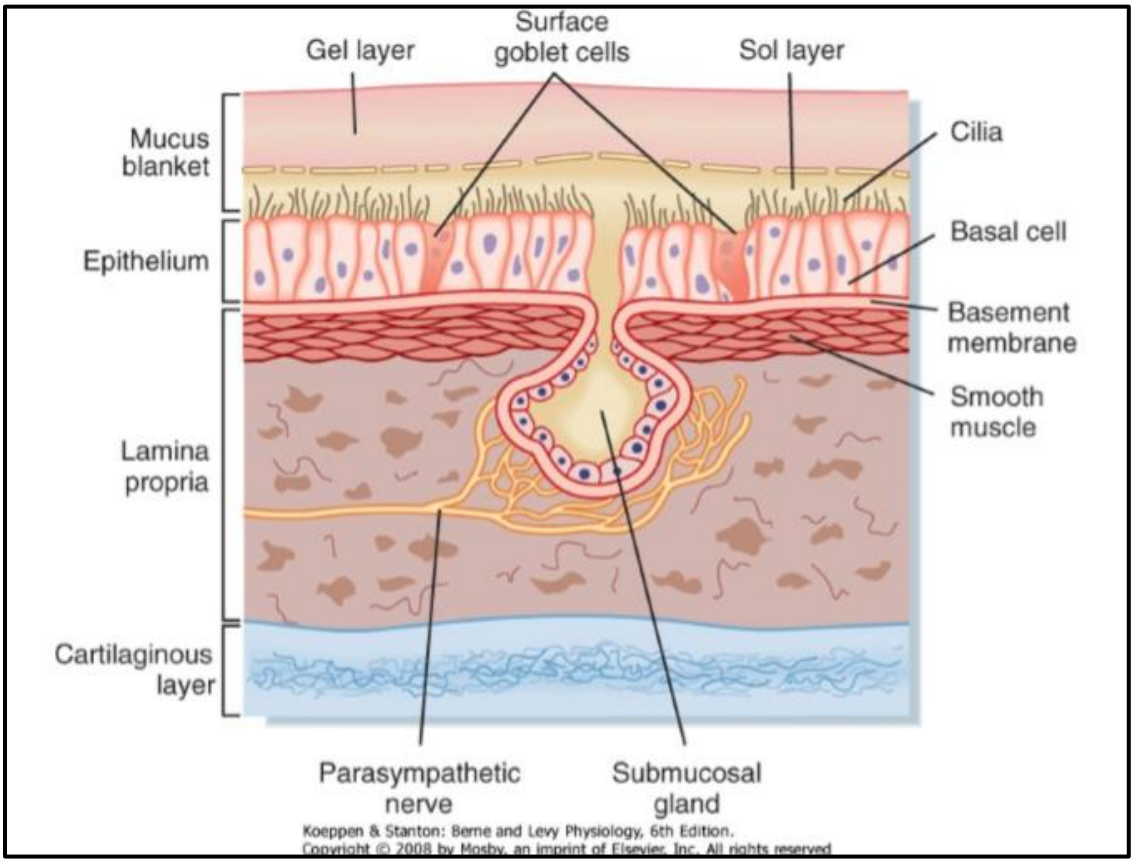


Figure 2: **The Mucociliary Clearance System**

Four cell types contribute to the quantity and composition of the mucus layer: goblet cells, mucous cells, and serous cells within the sub mucosal tracheobronchial glands, as well as Clara cells.

Goblet cells, also referred to as surface secretory cells, are present every five to six ciliated cells in the respiratory epithelium. They can be found up to the 5th tracheobronchial division and disappear beyond the 12th division. In many diseases, goblet cells appear further down the tracheobronchial tree, thus making the smaller airways more susceptible to obstruction by mucus plugging. Goblet cells secrete neutral and acidic glycoproteins rich in sialic acid in response to

chemical stimuli. In the presence of infection or cigarette smoke or in patients with chronic bronchitis, goblet cells can increase in size and number, and they secrete copious amounts of mucus. Injury and infection change the properties of the mucus secreted by goblet cells by increasing its viscosity.

Mucous cells, and serous cells within Sub mucosal tracheobronchial glands are present wherever there is cartilage in the upper regions of the conducting airways, and they secrete water, ions, and mucus into the airway lumen through a ciliated duct. Sub mucosal glands increase in number and size and can extend to the bronchioles in diseases such as chronic bronchitis (i.e., inflammation of the bronchi). This leads to increased mucus production, alterations in the chemical composition of the mucus (i.e., increased viscosity and decreased elasticity), and the formation of plugs that are manifested clinically as airway obstruction.

5) CILIA

There are approximately 250 cilia per airway epithelial cell, and each is 2 to 5 micronsin length. Cilia beat with a coordinated oscillation in a characteristic, biphasic, and wavelike rhythm called metachronism. They beat at approximately 1000 strokes/min, with a power forward stroke and a slow return or recovery stroke. During their power forward stroke, the tips of the cilia extend upward into the viscous mucus layer and thereby move it and the entrapped particles. On the reverse beat, the cilia release the mucus and withdraw

completely into the sol layer. Cilia in the nasopharynx beat in the direction that propels the mucus into the pharynx, whereas cilia in the trachea propel mucus upward toward the pharynx, where it is swallowed.

PARTICLE DEPOSITION AND CLEARANCE

PM10 (respirable particulate matter), PM2.5, PM1 and PM0.1 is defined as the fraction of particles with an aerodynamic diameter smaller than respectively 10, 2.5, 1 and 0.1 micron (for your information: 1 µm = 1 millionth of a meter or 1 thousandth of a millimetre). In comparison, the average diameter of a human hair equals 50-70 micron.

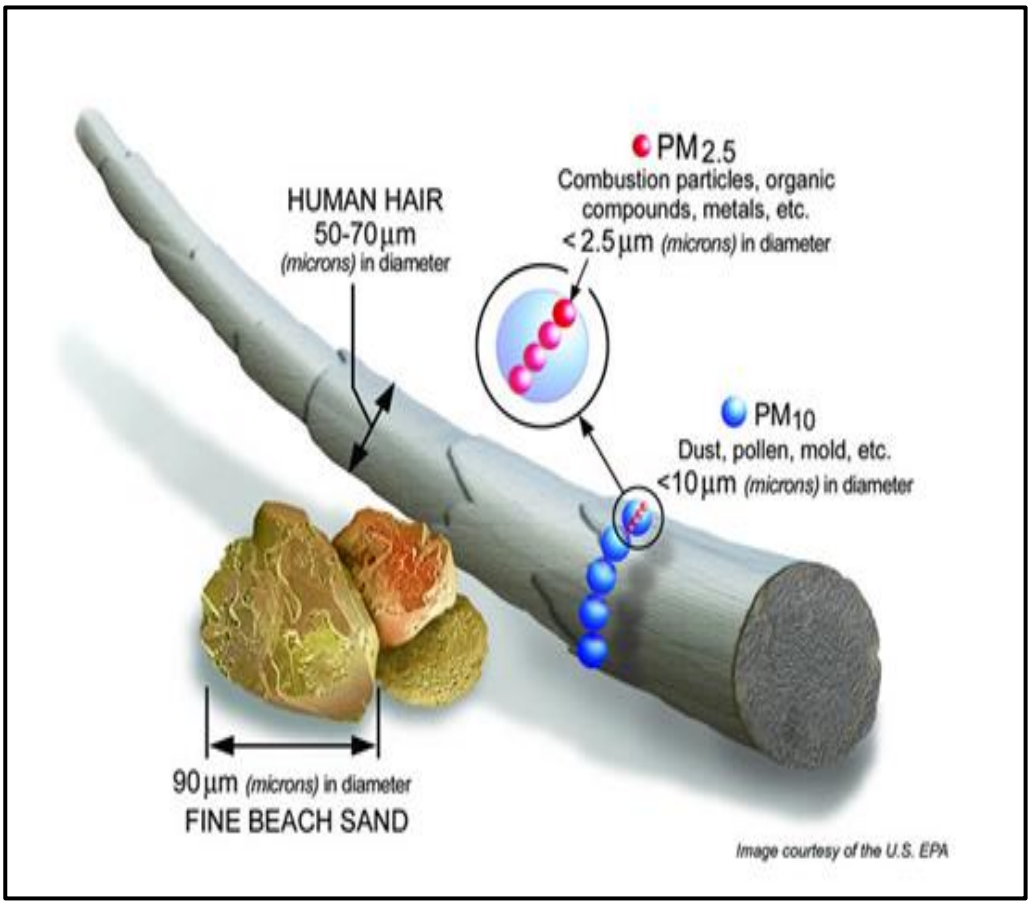


Figure 3: **Comparative Picture of Particulate Matter with Human Hair**

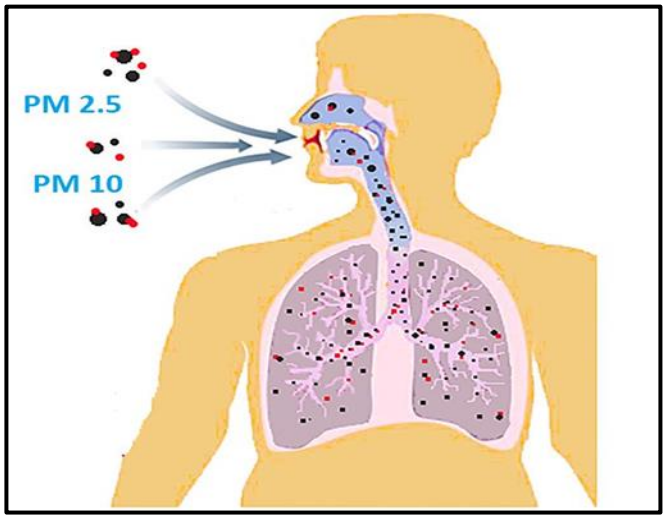


Figure 4: **PM Distribution in Lungs**

Deposition of particles in the lung depends on particle size and density, the distance over which the particle travels, and the relative humidity of the air.

Particles larger than 10 microns are deposited by impaction in the nasal passages and do not penetrate into the lower respiratory tract. Particles 2 to 10 microns in size are deposited in the lower respiratory tract predominantly by inertial impaction at points of turbulent flow (i.e., nasopharynx, trachea, and bronchi) and at airway bifurcations because their inertia (i.e., tendency to move in a straight direction) prevents them from changing directions rapidly.

In more distal areas, where airflow is slower, smaller particles (0.2 to 2 micron) are deposited on the surface by sedimentation secondary to gravity

Particles less than 0.2 micron are deposited by diffusion via brownian motion in the smaller airways and alveoli. The particle's diffusion coefficient is a major influence on the deposition of small particles. Thus, small particles can be

cleared only by lymphatic drainage or phagocytosis by alveolar macrophages. Macrophages migrate through the alveoli and engulf foreign or effete autologous materials in the airway lumen. Clearance of material by alveolar macrophages is usually rapid (<24 hours)[17].

6. IMMUNE DEFENSE SYSTEM

To deal with inhaled viruses, bacteria, and noxious agents, the respiratory system has developed specialized defence mechanisms that form the basis of the mucosal immune system in the lung. To avoid a continuous inflammatory state, which can cause lung damage, the lung must discriminate between what is harmful and what is not. Although inflammation is a protective response to injury or to an invading pathogen, inflammation usually disrupts the normal physiology. Accordingly, the lung has evolved "first-line" defence mechanisms that are designed to handle the offending agent with minimal or no inflammation. If the first-line defence mechanisms fail, an inflammatory response is initiated. The mucosa of the lung contains specialized adaptive immune cells (e.g., T lymphocytes with limited antigen recognition abilities and plasma cells that synthesize a non-complement-binding antibody, IgA) and innate immune cells (e.g., alveolar macrophages, natural killer cells, and dendritic cells) .These cells limit the immunological and inflammatory responses to foreign substances that enter the respiratory system.

7. MALT and BALT

The respiratory, gastrointestinal, and urinary systems are part of the body's mucosal immune system, which can function independently of the systemic immune system.

Hypersensitivity lung diseases are associated with an altered immune response to non-pathological organisms. It is not a typical allergic response in that symptoms arise 4 to 6 hours after contact with the inciting agent and eosinophils are not a prominent component. The lung pathology is more of a granulomatous like response with ensuing fibrosis.

The lung also has several unique defence features that limit airway inflammation. One of the specialized features is a unique antibody system that uses specialized functional features of the IgA antibody. In sub mucosal areas, plasma cells synthesize and secrete IgA, which migrates to the sub mucosal surface of epithelial cells, where it binds to a surface protein receptor, poly-Ig. The poly-Ig receptor aids in pinocytosis of IgA into the epithelial cell and eventual secretion (exocytosis) of IgA into the airway lumen. During exocytosis of the IgA complex, the poly-Ig is enzymatically cleaved, and a portion of it, the secretory piece, is still associated with the complex. The secretory piece remains attached to the IgA complex in the airway, and it helps protect the IgA complex from proteolytic cleavage in the lumen. The IgA-antibody system is very effective in binding particulates and viruses before they invade epithelial cells,

and it aids in removal of these substances through the mucociliary clearance system. The IgA-antigen immune complex does not bind complement in the same classic manner as other immune complexes do; this limits its proinflammatory properties.

TCRγδ cells are the "first line of defence" of epithelial surfaces, and they prevent the development of inflammation mediated by antigen-specific T cells. These cells provide a bridge between adaptive and innate immunity. TCRγδ cells also suppress the IgE response to inhaled antigen.

Resident populations of functionally active NK cells are present in the lung interstitium. NK cells are a major component of the body's innate immune defence system against invading pathogens such as herpes-viruses and various bacterial infections

8. Dendritic Cells and Alveolar Macrophages

Dendritic cells and alveolar macrophages are the first nonepithelial cells to respond to a foreign substance. If the foreign material stays within the air space in the lower respiratory system (alveolar ducts and alveoli), it will be phagocytized by alveolar macrophages and removed by the lymphatic system. However, if it penetrates and reaches the interstitial areas, it will come in contact with dendritic cells. Dendritic cells capture, process, and present antigen to T cells, as well as activate or suppress the T cell response.

9. Toll-like Receptors (TLR)

Because most inhaled substances are non-pathogenic, the body has developed a recognition system to identify potentially harmful pathogenic substances. TLR-4 is specific for the gram-negative bacterial product lipopolysaccharide, whereas TLR-2 is specific for lipoproteins associated with gram-positive bacteria. In the lung, bronchial epithelial cells and alveolar type II epithelial cells express TLR-2 and TLR-4. Macrophages and dendritic cells in the lung and other organs also express TLRs. Thus, in addition to classic phagocytic cells, bronchial and alveolar epithelial cells play active roles in host defence via the PAMP-TLR recognition system.

MECHANICS OF VENTILATION

Inspiration is the active phase of breathing; the muscles of the chest wall, mainly the diaphragm, (external intercostal muscles, sternocleidomastoid muscle, serratus anterior muscle, scalene muscle) contract and move down into the abdomen, thereby resulting in negative pressure inside the chest. Gas then flows from higher to lower pressure. The intra pleural pressure becomes more negative from -2.5mm Hg to -6mmHg during inspiration. This is created by expansion of chest wall which pulls the lung along with it with such great force. The surface tension-reducing and anti-stick properties of surfactant diminish the work of breathing and help stabilize alveoli.

Expiration is passive during quiet breathing. The muscles of inspiration relax. The chest wall is pulled back to its original position due to elastic recoiling of lungs. The intra pleural pressure becomes positive and air is exhaled.

At the end of expiration,the recoiling force of lungs and thoracic cage balance each other.The intra pleural pressure becomes -2.5 mm Hg[15].

LUNG MECHANICS is the study of the mechanical properties of the lung and the chest wall (which includes the rib cage, diaphragm, abdominal cavity, and anterior abdominal muscles). An understanding of lung mechanics is important to comprehend both how the lung works normally and how the lung works in the presence of disease because almost all lung diseases affect the mechanical properties of the lung. Lung mechanics includes static mechanics (the mechanical properties of a lung whose volume is not changing with time) and dynamic mechanics (properties of a lung whose volume is changing with time). **[STATIC & DYNAMIC LUNG MECHANICS]**

I. STATIC LUNG MECHANICS

LUNG VOLUMES AND CAPACITIES

Lung volumes are determined by the balance between the lung's elastic properties and the properties of the muscles of the chest wall.

1. Tidal volume is the volume of air inspired or expired during quiet breathing (500ml)

2. Inspiratory reserve volume is the amount of air inspired with maximum inspiratory effort above the normal tidal volume (3000ml).

3. Expiratory reserve volume is the volume of air expired with maximum expiratory effort after the normal tidal expiration: (1100ml)

4. Residual volume is the volume of air remaining in the lungs after the forceful expiration (1200ml)

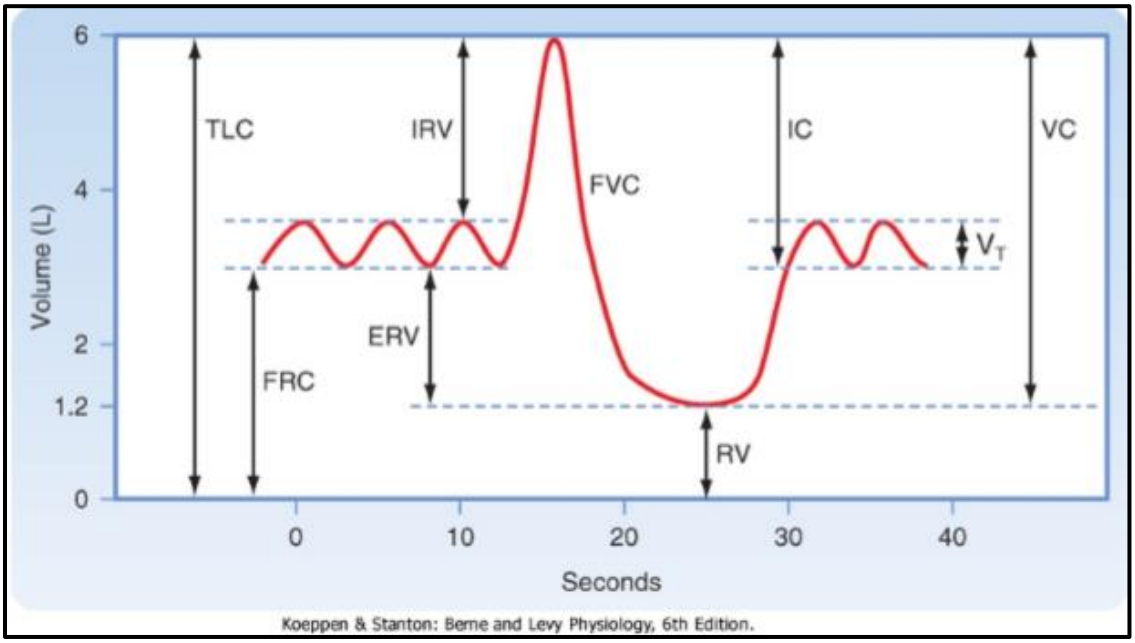


Figure 5: **Lung Volumes and Capacities**

The pulmonary capacities are

1. **Inspiratory capacity -** The maximum amount of air inspired after completing the tidal expiration (3500ml).

2. The **functional residual capacity**- The amount of air remaining in the lung at the end of normal expiration (2300ml)

3. The **vital capacity** - The maximum amount of air expired forcefully after a maximum inspiratory effort (4600ml)

4. The **total lung capacity**- The volume of air present in the lung after a maximum inspiration (6 litres)

Table 2: PFT Indices

Indices based on Volume	Indices based on Time
FVC	FORCED EXPIRATORY TIME (FET)
FEV1	
FEV1 / FVC ratio (or) FEV1%	
FEF 25-75%	
MAXIMUM VOLUNTARY VENTILATION (MVV)	
SLOW VITAL CAPACITY	
PEAK EXPIRATORY FLOW (PEF)	

INDICES BASED ON VOLUME

1) FORCED VITAL CAPACITY (FVC)

The maximum volume of air expired forcefully and rapidly after a maximal inspiration is FVC. It equals VC ; FVC and VC should be within 200ml of each . FVC is < 80% of predicted value is abnormal. Low FVC is a non-specific. FVC may be low in both obstructive and restrictive disorder. But in restrictive disorder FVC is too low compared to FEV1.

2) FEV1

The volume of air expired in first second of an FVC manoeuvre. < 80% of predicted value is considered abnormal. FEV1 is low in obstructive and restrictive disorders, but in obstructive disorder FEV1 is considerably low when compared to FVC.

3) FEV1 / FVC ratio (or) FEV1%

The FEV1 expressed as a percentage of FVC. Normal value - 70% FEV1% = (FEV1/FVC) × 100

4) FEF 25-75%

Forced expiratory flow over the middle half of FVC manoeuvre. Indicates the status of medium to small airways. Normal value is4 to 5 litres per second that is 65% of predicted value.

5) MAXIMUM VOLUNTARY VENTILATION (MVV)

The maximum volume of air expired over a specified period of time (12 sec for normal subjects). Airway resistance, respiratory muscle, compliance of the lung and chest wall andventilatory control mechanisms influences MVV. Normal values - 150 – 200 L/min in healthy young men. Decreased in patients with moderate to severe obstructive disease. MVV may be normal in patients with restrictive pulmonary disease.

6) SLOW VITAL CAPACITY

Slow vital capacity is the volume of air measured from a slow, complete expiration following a maximal inspiration, without forced or rapid effort .

7) PEAK EXPIRATORY FLOW (PEF)

The maximal expiratory flow achieved during a maximum forced expiration starting at total lung capacity. Patient effort is indicated by PEF during spirometry. Large airway function is measured by PEF. Home monitoringfor asthma patients is done with this parameter [18, 21, 22].

INDICES BASED ON TIME

FORCED EXPIRATORY TIME (FET)

The time taken to expire a specified portion of the forced vital capacity is known as forced expiratory time (FET). If it is > 4 second it indicates some degree of airflow obstruction.

DYNAMIC LUNG MECHANICS

The principles that control air movement into and out of the lung. Dynamics is that aspect of mechanics that studies physical systems in motion. Airflow in Airways - Two major factors determine the speed at which gas flows into the airways for a given pressure change: the pattern of gas flow and the resistance to airflow by the airways. , the major site of resistance along the bronchial tree is the large bronchi. The smallest airways contribute very little to

the overall total resistance of the bronchial tree The reason for this is twofold. First, airflow velocity decreases substantially as the effective cross-sectional area increases (i.e., flow becomes laminar). Second and most important, the airway generations exist in parallel rather than in series. The resistance of airways in parallel is the inverse of the sum of the individual resistances; therefore, the overall resistance of the small airways is very small.

PULMONARY FUNCTION TESTS

Pulmonary function tests are age old, still valid and very important tests for assessing the functions of respiratory system. They aid to knowledge about the clinical condition, diagnosis and prognosis of a disease. Normally a person attains maximal lung function around 20 to 25 years of his / her age. After 30 to 35 years of his / her age there is decline in lung function. The lung function decline to a moderate extent even before clinical symptoms and signs develop. So the assessment of severity of disease is difficult with symptoms and signs alone, which may lead to inadequate treatment and control of disease. So early lung function test measurement is very important for early intervention and control, and also to monitor the progress of the disease [18,19].

The factors that determine the ability of lungs to exchange gases effectively are:

Factors contributing for ventilation

1) The diaphragm and other thoracic muscles creating a sub atmospheric pressure.

2) The patent airways allowing the gas to reach the alveoli.

Factors determining the diffusion and perfusion of lungs

1) The intact and effective respiratory membrane for the diffusion of oxygen and carbon dioxide.

2) The normal functioning of cardiovascular system providing adequate blood supply to the lungs.

The pulmonary function tests provide valuable information about all the above processes of ventilation, diffusion and perfusion[(20)].

Based on the aspects of lung function they measure, the pulmonary function tests are categorized as:

1) Airway function test – VC, FVC, FEV1, PEFR, FEF
2) Lung Volume and Ventilation Test – FRC, TLC, Minute ventilation
3) Diffusion Capacity Test – DLCO
4) Blood Gas and Gas Exchange Test – ABG, pulse oximetry, capnography
5) Cardiopulmonary Exercise Test – Test with exhaled gas analysis, Test with blood gas analysis

6) Metabolic Measurement – resting energy expenditure, substrate utilisation.The airway function and lung volume are measured with spirometry [19].

SPIROMETRY

Spirometry is a basic, easiest but powerful tool that can detect and differentiate the lung disorders and is also used as a tool for follow up of patients with pulmonary disorders. It is very useful in determining the pattern of lung dysfunction. False positive results may occur if not performed properly. Measurement of expiratory flow rates and expiratory volumes is an important clinical tool for evaluating and monitoring respiratory diseases. Commonly used clinical tests have the patient inhale maximally to TLC and then exhale as rapidly and completely as possible to RV. The test results are displayed either as a spirogram or as a flow-volume curve/loop.

Results from individuals with suspected lung disease are compared with results predicted from normal healthy volunteers. Predicted or normal values vary with age, gender, ethnicity, height, and to a lesser extent, weight Abnormalities in values indicate abnormal pulmonary function and can be used to predict abnormalities in gas exchange. These values can detect the presence of abnormal lung function long before respiratory symptoms develop, and they can be used to determine disease severity and the response to therapy.

STANDARDIZATION OF SPIROMETRY

According to American Thoracic Society the spirometers are standardized as follows:

1. It should record at least FVC and FEV1.
2. It should record a flow volume curve or a flow volume loop or both.
3. It should be able to measure up to 15 seconds for FVC.
4. It should have a capacity of 8 litres.
5. It should measure volume within < 3% error or within 0.05 litres of a reference value whichever is greater.
6. It should measure flow within <5% error or 0.2 litres per second whichever is greater.
7. The values given by spirometer are corrected for body temperature, ambient pressure and saturated with water vapour (BTPS).
8. It can be calibrated with a 3 litres syringe[21,22].

INDICATIONS FOR DOING SPIROMETRY

1) Detectif any lung dysfunction is present or absent
2) Assessing severity of lung disease
3) Monitoring disease progression
4) Assess the treatment efficacy
5) Measure the effects of occupational and environmental exposure of air pollutants.

6) For pre op assessment.

7) For impairment or disability quantification [23,24].

CONTRAINDICATIONS FOR DOING SPIROMETRY

1) Respiratory infections

2) Recent myocardial infarction within 1 month prior to the procedure.

3) Unstable cardiovascular status.

4) Haemoptysis

5) Pneumothorax

6) Recent surgeries of eye / thorax / abdomen

7) Stress incontinence.

8) Dementia or confused patient.

9) Oral or facial pain exaggerated by the mouth piece[23,24].

RECORDING OF SPIROMETRY

Graphical and numerical recordings are made. Graphically as;

1) Spirogram – volume versus time graph

2) Flow rate versus volume

Recordings are done as;

a) Flow volume curve when only expiratory flow is recorded

b) Flow volume loop when both expiratory flow and inspiratory flow is recorded [14,19].

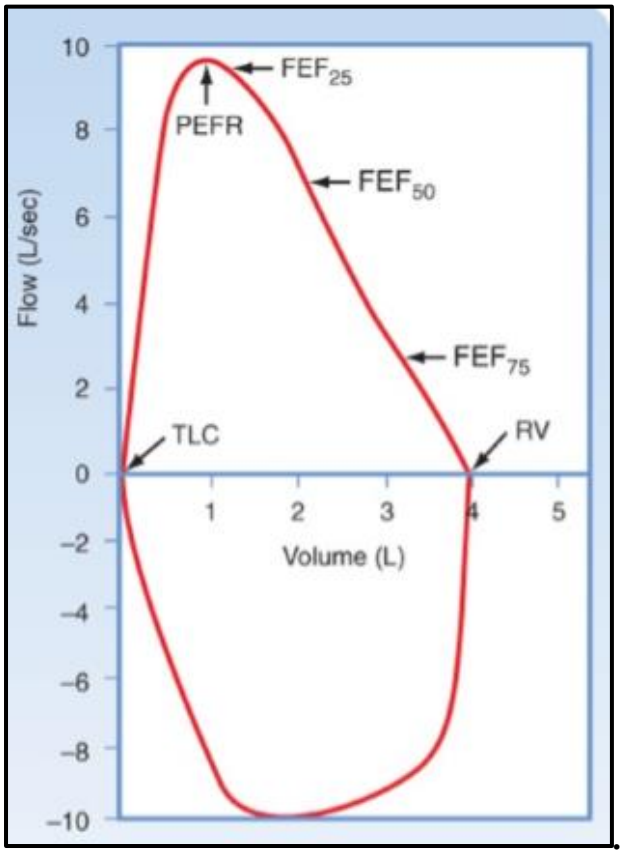


Figure 6 : Flow – volume loop

A flow-volume curve or loop is created by displaying the instantaneous flow rate during a forced manoeuvre as a function of the volume of gas. This instantaneous flow rate can be displayed both during exhalation (expiratory flow-volume curve) and during inspiration (inspiratory flow-volume curve) Expiratory flow rates are displayed above the horizontal line and inspiratory flow rates below the horizontal line. The flow-volume loop yields data for three main pulmonary function tests: (1) the FVC; (2) the greatest flow rate achieved during the expiratory manoeuvre, called the peak expiratory flow rate (PEFR), and (3) expiratory flow rates. When the expiratory flow-volume curve is divided into quarters, the instantaneous flow rate at which 50% of the VC remains to be

exhaled is called the FEF50 (also known as the Vmax50), the instantaneous flow rate at which 75% of the VC has been exhaled is called the FEF75 (Vmax75), and the instantaneous flow rate at which 25% of the VC has been exhaled is called the FEF25 (Vmax25).

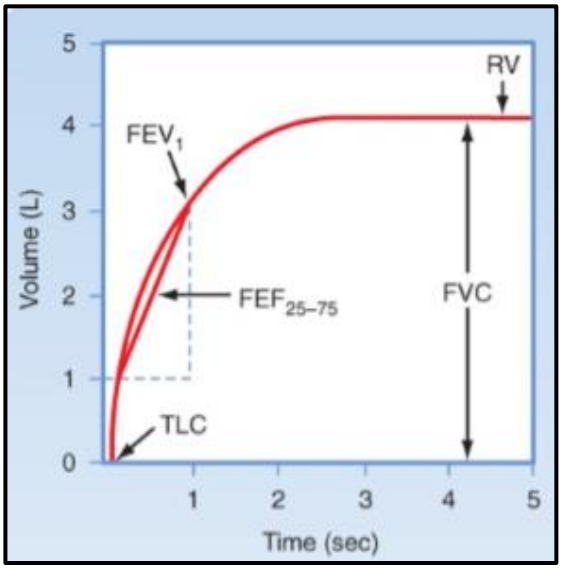


Figure 7: Volume vs time graph (Spirogram)

A Spirogram displays the volume of gas exhaled against time FVC, FEV1, FEV1/FVC, FEF 25-75%. Vertical axis represents flow and horizontal axis represents volume. Upward flow is expiration and downward flow is inspiration. Peak flows for expiration and inspiration (PEF and PIF) are also shown and the instantaneous flow (FEF) at any point in the FVC also can be measured directly.

PATHOGENESIS OFLUNG DISEASES

Table 3: Classification of Lung Disease

LUNG DISEASES				
OBSTRUCTIVE DISEASES			**RESTRICTIVE DISEASES**	
COPD	Bronchiectasis	Bronchial Asthma (Reversible)	Chronic interstitial and infiltrative diseases	Chest wall disorders
e.g.,			e.g.,	**e.g.,**
1. Chronic bronchitis			1.Pneumoconioses	1.Neuromuscular diseases
2.Emphysema			2.Interstitial fibrosis of unknown etiology	2. Pleural diseases

Obstructive lung diseases (or airway diseases) are characterized by an increase in resistance to airflow due to partial or complete obstruction at any level from the trachea and larger bronchi to the terminal and respiratory bronchioles. These are contrasted with restrictive diseases, which are characterized by reduced expansion of lung parenchyma and decreased total lung capacity[(25)].

1. OBSTRUCTIVE LUNG DISEASE

In obstructive airway disease less air flows in and out of airways due to one of the following reasons:

1. Airways& air sacs lose their elasticity due to inflammation of airways,
2. Bronchial hyper reactivity leading to air sac wall destruction,

3. Walls of the airways become thick and inflamed,

4. Airways make more mucus which clog them.

5. Increased mucus production, decreased ciliary action and loss of elasticity of bronchial wall leading to obstruction of airways.

Table 4: Obstructive Lung Diseases

Clinical Term	Anatomic Site	Major Pathologic Changes
Chronic bronchitis	Bronchus	Mucous gland hyperplasia, hypersecretion
Bronchiectasis	Bronchus	Airway dilation and scarring
Asthma	Bronchus	Smooth muscle hyperplasia, excess mucus, inflammation
Emphysema	Acinus	Airspace enlargement; wall destruction
Small-airway disease, bronchiolitis*	Bronchiole	Inflammatory scarring/obliteration

2. RESTRICTIVE LUNG DISEASE

Normally interstitial space has minimal connective tissue, extracellular matrix and minimal inflammatory cells in them. This allows efficient gas exchange between alveoli and capillaries. If any inflammation of interstitium occurs, the lungs responds to the damage and try to repair the damage. If the inflammation persists or if there is imperfect repairing process occurs, then this may lead to permanent damage of lung parenchyma resulting in restrictive lung disease [(25)].

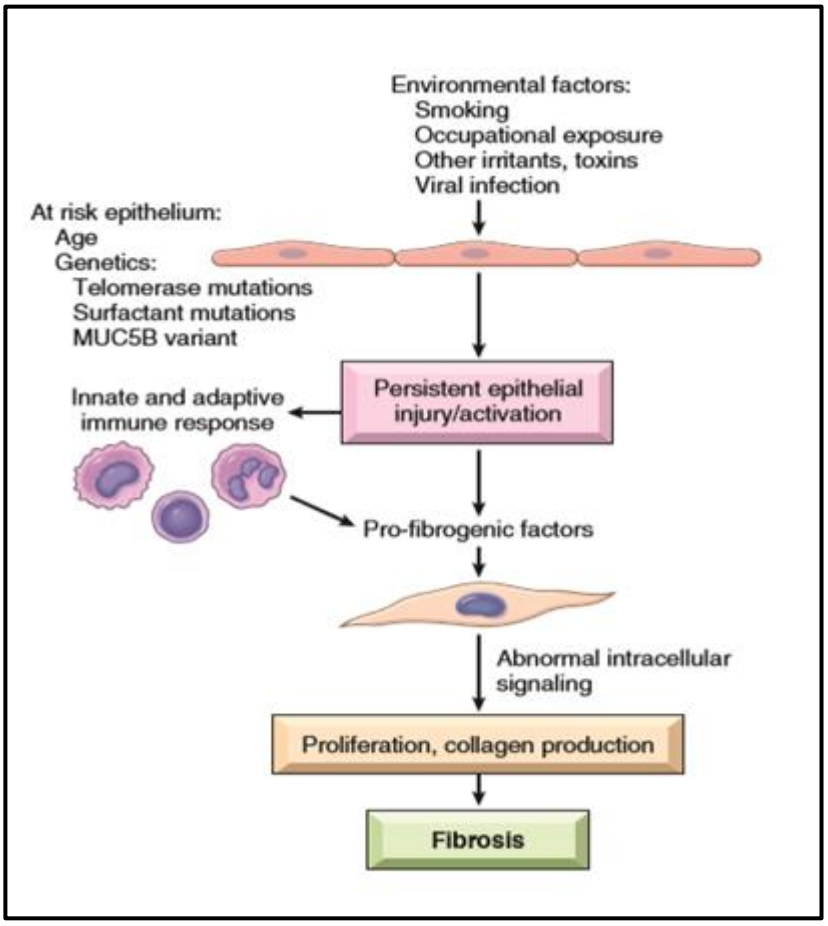


Figure 8: Pathogenesis of Restrictive Lung Disease

The distinction between these chronic non-infectious diffuse pulmonary diseases is based primarily on pulmonary function tests.

SPIROMETRY PATTERN IN LUNG DISEASES

NORMAL

FEV1 and FVC >80% predicted value

FEV1 / FVC ratio >70% of predicted value is considered normal.

Obstructive lung disorders

- FEV1 <80% of predicted value

- FVC normal or reduced (if reduced usually to a lesserdegree than FEV1)
- FEV1 / FVC ratio <70% of predicted value .

Restrictive lung disorder

- FVC <80% of predicted value
- FEV1 normal or reduced (if reduced usually to a lesserdegree than FVC)
- FEV1 / FVC ratio 70% or > 70% of predicted value .

Mixed function disorder (both obstructive and restrictive)

- FVC and FEV1 <80% of predicted value
- FEV1 / FVC ratio <70% of predicted value [31].

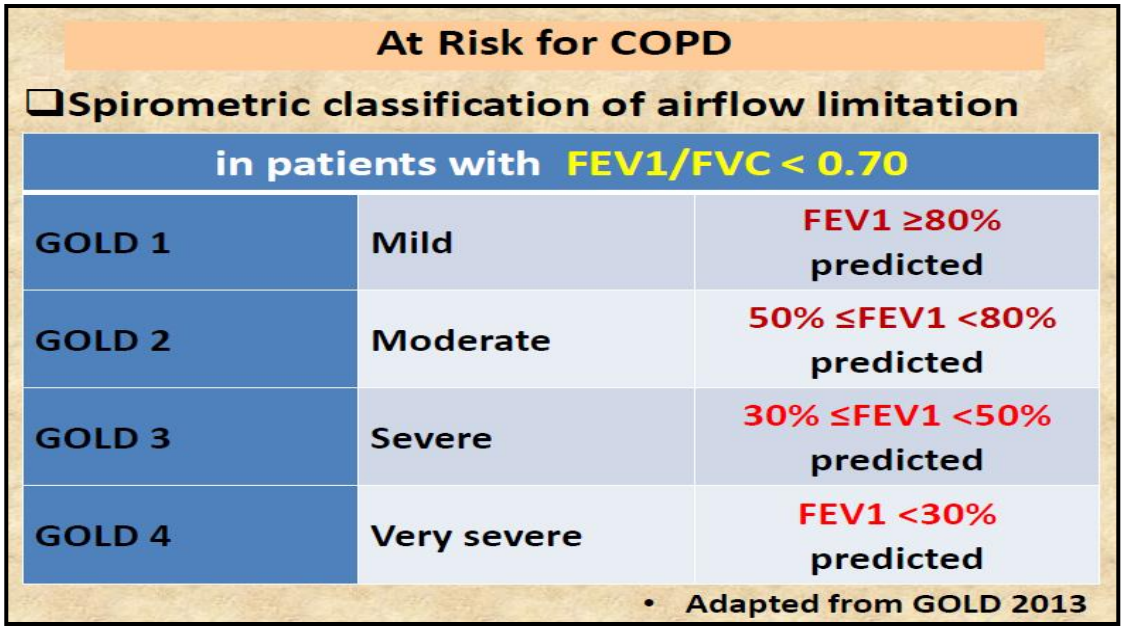


Figure 9: GOLD CRITERIA 2013 [26]

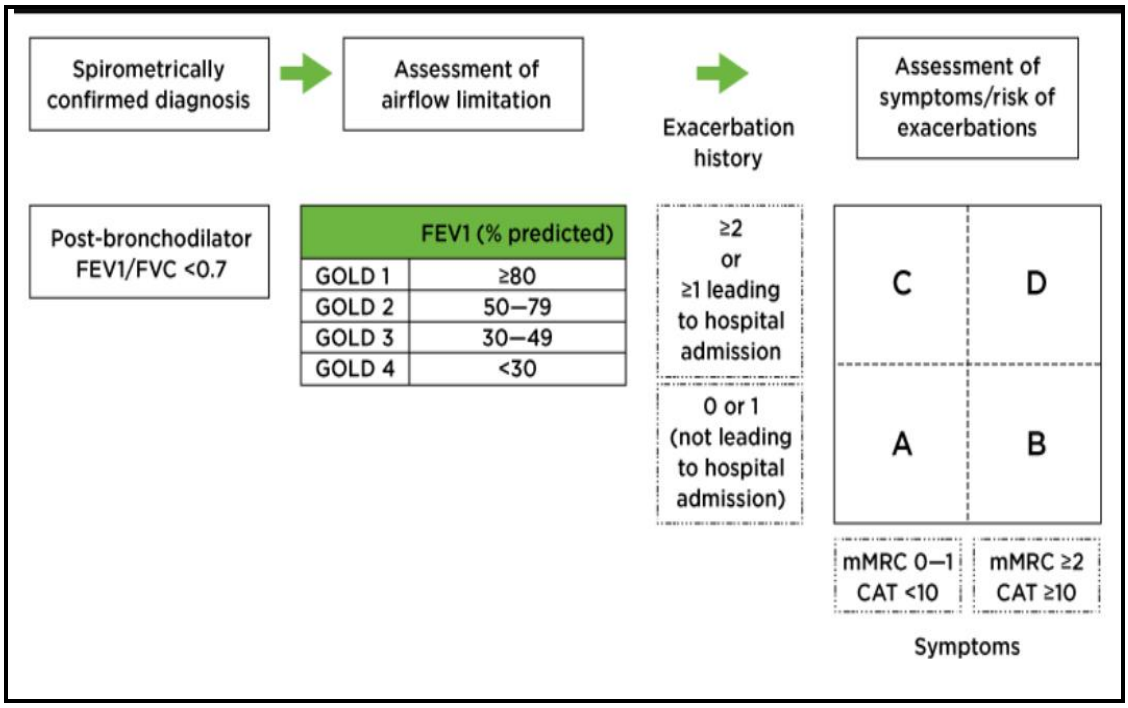


Figure 10: GOLD CRITERIA 2017[26]

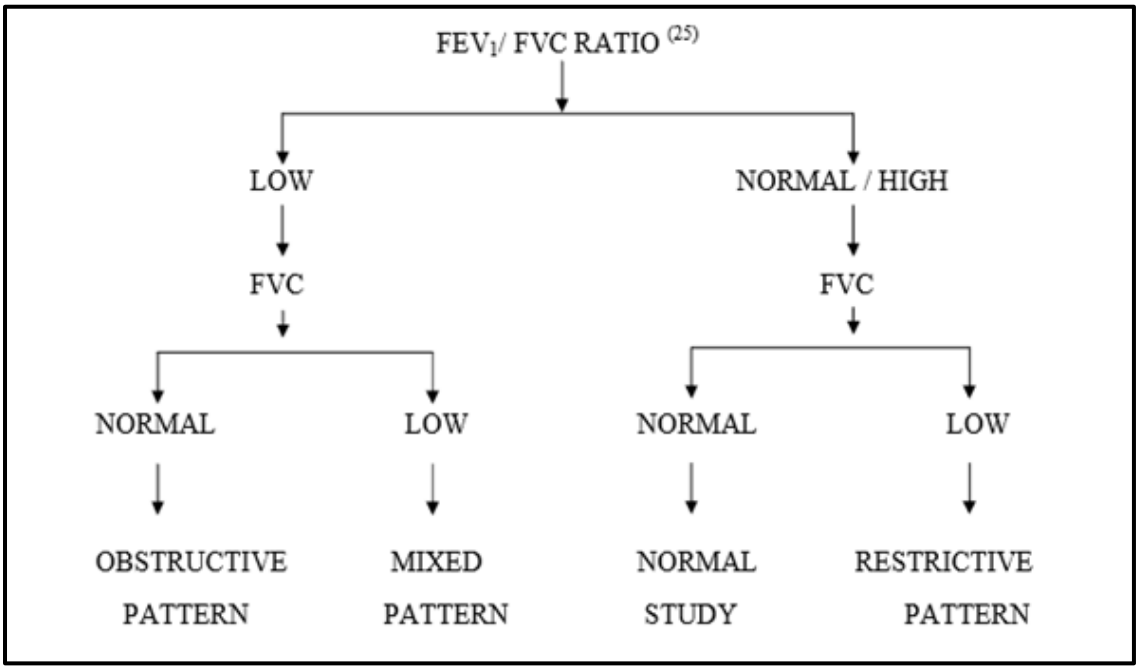
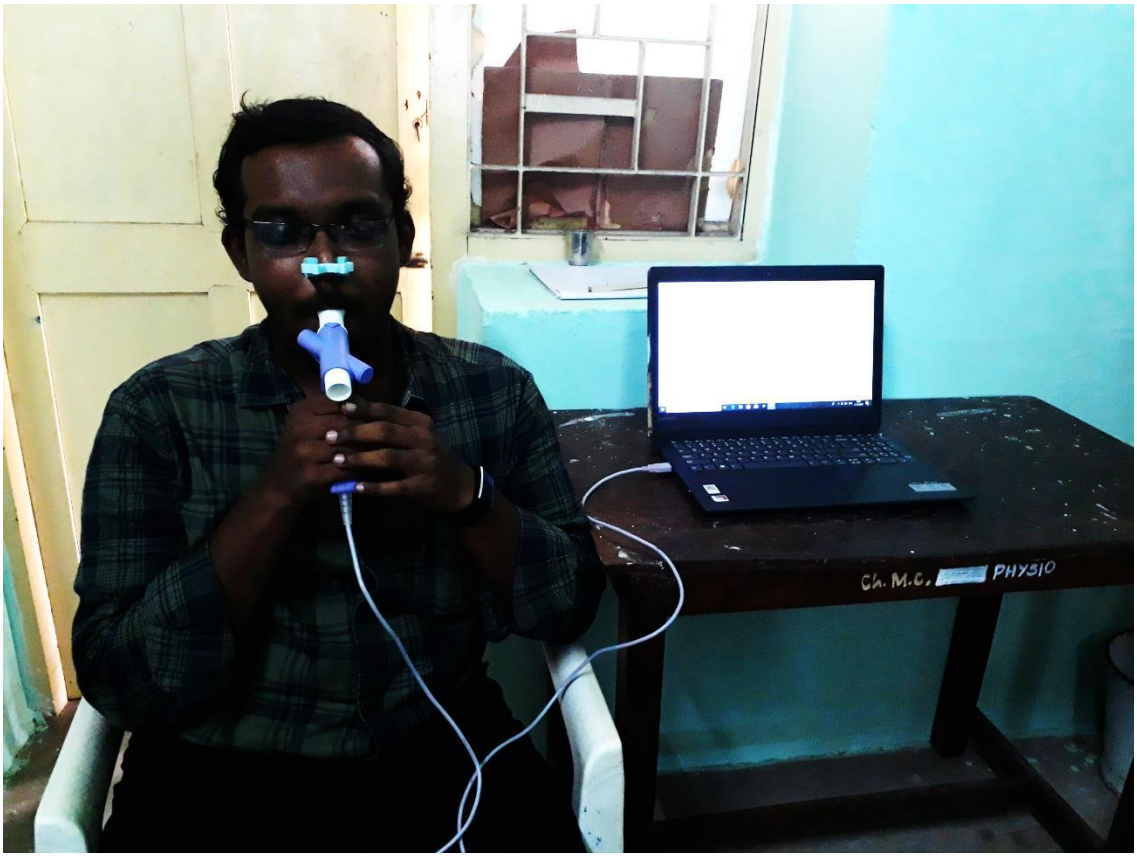


Figure 11: Interpretation of Patterns of Lung Function Impairment


Ch. M.C. PHYSIO

REVIEW OF LITERATURE

Employment in hospital sanitary service was found to be associated with increased respiratory symptoms and decline in some of the pulmonary function parameters.

The global and Indian scenario in solid waste disposal and hospital waste disposal has been highlighted earlier. **ASSOCHAM** Associated Chambers of Commerce and Industry of India) and Velocity joint study in their study – "Unearthing the growth curve & necessities of BMW management in India – 2018" has said ,India is likely to generate about 775.5 tonnes of medical wastes/day by 2022 from current level of 550.9 tonnes/day[27].

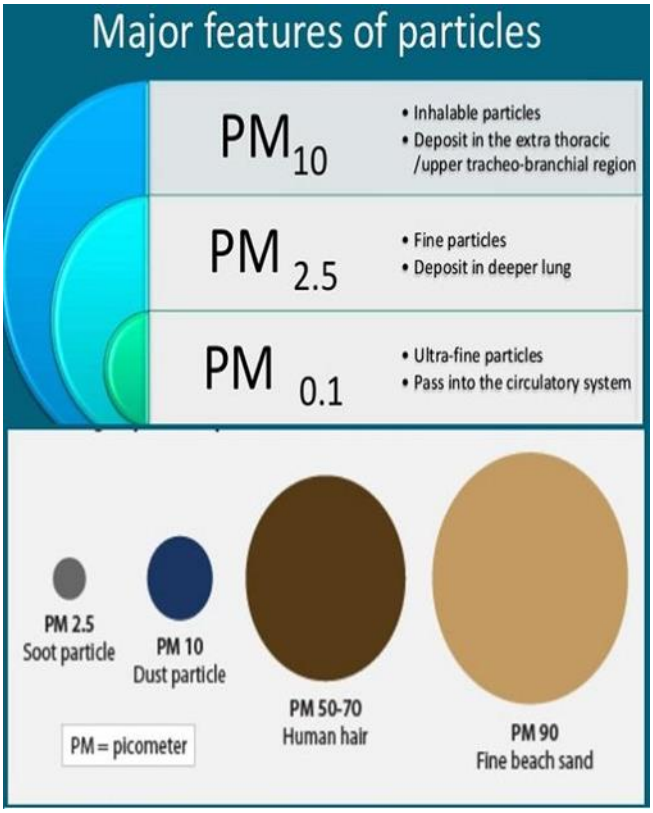
The sanitary workers are the first person to handle this, risking their health status. A variety of ailments occur involving all systems, most commonly the respiratory system, as lung is in direct contact with external environment.

PrabhakumariChellamma et al., 2015, cross sectional morbidity study was conducted among 601 sanitation workers corporation in Thrissur, Kerala, India, highlighted that these workers suffer from skin diseases, respiratory and gastrointestinal problems, eye and ear infections and accidental injuries [28].

ETIOLOGY (Particulate matter, Bio aerosols)

Sean H Ling et al., 2009 in their study showed that inhalable particulate matter (PM10) shows a strong association with adverse respiratory health effects,

even when adjusted for other major risk factors such as cigarette smoking. PM is a mix of solid or liquid particles suspended in the air. PM is deposited at different levels of the respiratory tract, depending on its size: coarse particles (PM10) in upper airways and fine particles (PM2.5) can be accumulated in the lung parenchyma, inducing several respiratory diseases. PM can be constituted by organic, inorganic, and biological compounds. All these compounds are capable of modifying several biological activities, including alterations in cytokine production, coagulation factors balance, pulmonary function, respiratory symptoms, and cardiac function. It can also generate different modifications during its passage through the airways, like inflammatory cells recruitment, with the release of cytokines and reactive oxygen species .These inflammatory mediators can activate different pathways, such as MAP kinases, Nuclear Factor -κB, and Stat-1, or induce DNA adducts. All these alterations can mediate obstructive or restrictive respiratory diseases like asthma, COPD, pulmonary fibrosis, and even cancer. The PM, as opposed to gases such as nitrogen dioxide and ozone, are the strongest associated with increased mortality[17].



Agarwal S et al., 2016, Size distribution analysis shows that bacteria were mostly abundant in fine particle sizes, i.e. <0.43-2.1 microns, but few peaks were also observed in size ranges between 5.8->9.0 microns. Fungal spores mostly peaked in coarse sizes (2.1-5.8 microns) and showed unimodal size distribution. Predominant identified bacterial strains were mostly belonged to Bacillus, Staphylococcus, Streptococcus, Klebseilla and Escherichia genera. Most of the identified fungal spores are known for adverse health effects causing numerous allergic and pathogenic inflammations. These results suggest that the open-solid waste dumping sites are a major source of bio aerosols, and residents living in the nearby areas of landfills are at high health risks[(29)].

Sangolli et al., 2018, Chronic exposure to air pollution and ambient particulate matter (PM) has been found to be associated with increased rates of hospitalization and mortality due to respiratory illnesses. Short term exposure to PM is being found to be associated with impaired FEV1 Sweeping with brooms, vehicular movements and other human activities, raise the dust particles in the air [30–32].

Chestnut LG et al., 1991, The relationship between pulmonary function and quarterly average levels of total suspended particulates (TSP) was examined for adults who resided in 49 of the locations where the First National Health and Nutrition Examination Survey (NHANES I) was conducted. Statistically significant relationships were observed with FVC and FEV1and TSP levels. These relationships remained strong across several specifications and sample changes, e.g., exclusion of cities with two highest and two lowest TSP levels, restriction of sample to whites only. The results indicate a 1 standard deviation increase (about 34 $\mu g/m^3$) in TSP from the sample mean of 87 $\mu g/m^3$ was associated with an average decrease in FVC of 2.25%. The results of this analysis also suggest that there is a threshold level (i.e., approximately 60 $\mu g/m^3$ [quarterly average]) of TSP below which a relationship with pulmonary function ceases to exist[33].

BIO-AEROSOLS

Shadab et al., 2013, in their study says dust particles of larger size are either swallowed or coughed out but the smaller particles between 1-5 micrometres settle down in the smaller bronchioles as a result of gravitational precipitation. Particles smaller than 1 micron in diameter diffuse into the alveoli and adhere to alveolar fluid which are then taken up by alveolar macrophages which later on leads to tissue destruction.

J. Douwes et al., 2002, In their review article says,Bio aerosols or organic dust may consist of pathogenic or non-pathogenic live or dead bacteria and fungi, viruses, high molecular weight allergens, bacterial endotoxins, mycotoxins, peptidoglycans, β(1→3)-glucans, pollen, plant fibres, etc. Nowadays it is recognized that exposures to biological agents in both the occupational and residential indoor environment are associated with a wide range of adverse health effects with major public health impact, including contagious infectious diseases, acute toxic effects, allergies and cancer. Workers in this industry (e.g. waste sorting, organic waste collection and composting) are often exposed to very high levels of microorganisms[34–36].

Hansen et al., 1993, Bio aerosols generated by decaying organic waste, vehicle exhaust fumes and bad weather conditions may all contribute to respiratory problems. Bio aerosols contain several agents capable of inducing inflammation in the airways[37].

Jurgen Bunger et al., 2000, in a cross sectional study, work related health complaints and diseases of 58 compost workers and 53 bio waste collectors were investigated and compared with 40 control subjects. Levels of specific IgG antibodies to moulds and bacteria were measured as immunological markers of exposure to bio aerosols.Increased antibody concentrations against fungi and actinomycetes were measured in workers at composting plants. The concentrations in bio waste collectors did not differ significantly from those in the control subjects [38, 39].

ENDOTOXINS

Ivens et al., 1997, in their severalstudies have shown the effects of chronic exposure to wastes on respiratory function and related it to the high dust levels, micro-organisms, fungal spores and endotoxins [40].

J. Thorn and R. Rylander1998., performed a study on 21 healthy subjects who were made to inhale 40 µg lipopolysaccharide and were examined before and 24 hours after exposure , to assess the usefulness of the induced sputum technique for evaluating the presence of airways inflammation using inhaled endotoxin (lipopolysaccharide) as the inducer of inflammation. There is increasing evidence that diseases caused by exposure to bio aerosols are mainly of a non-allergic inflammatory nature. The results supported previous studies that inhaled endotoxin causes an inflammation at the exposure site itself, as well as general effects[34].

T Singh, Mrs OnnicahMatuka, M Jeebhay in Current Allergy and Clinical Immunology 2010, says Endotoxin, a by-product of Gram-negative bacteria found ubiquitously in the environment. Workers in different occupational settings are exposed to organic dust containing endotoxins and are at risk of developing respiratory diseases. The relationship between endotoxin exposure and health effects is still controversial because some studies have demonstrated protective response for developing asthma, while others show priming of the allergic response and an exacerbation of asthma. Endotoxins acting on their own cause neutrophilic inflammatory responses and a decline in lung function in exposed workers, acting as a natural adjuvant to augment atopic inflammation and asthma [(41)].

Ekram W. Abd El-Wahab et al., 2014, in their study on 346 workers different solid waste management activities regarding personal hygiene, the practice of security and health care measures showed high prevalence of gastrointestinal, respiratory, skin and musculoskeletal morbidities.High dust levels, micro-organisms, fungal spores and endotoxins[(33,34,40,42–46)] or the presence of higher levels of total suspended particulate matter does impair lung function on chronic exposure. Breathlessness may be due to air way obstruction and inflammation.

Kozajda A et al., 2009 ,in their study showed fungal air contamination with *Aspergillusflavus*, *Aspergillusfumigatus*, and *Stachybotryschartarum* species which cause pulmonary disease[(47,48)].

Nielsen et al.,1997, a Danish studies on waste collectors bioaerosol exposure showed generally the median exposure levels ranged from 10^5 to 10^6 cells m^{-3} (total microorganisms), 10^4 to 10^5 cfu m^{-3} (culturable fungi) and 10^3 to 10^4 cfu m^{-3} (culturable bacteria). The type of waste was a governing factor for exposure. Garden waste collectors frequently experienced concentrations exceeding 10^5 cfu m^{-3} for mesophilic fungi and 10^4 cfu m^{-3} for the thermophilic fungus *Aspergillusfumigatus*. Workers collecting compostable, mixed and sorted waste occasionally experienced similar concentrations of the fungal groups while workers collecting 'bulky waste' and paper had low exposure. Type of collection vehicle was identified as another governing factor for exposure.

Vehicles loaded from the top (approximately 3 m above the ground) caused lower exposure (by a factor of 25) to fungi than vehicles loaded at the level or the breathing zone of the workers.

Exposure was also affected by season of the year—the concentration of total microorganisms, culturable fungi, *Aspergillusfumigatus* and endotoxin was low in winter. Likewise, dust may also be used as an indicator of exposure to total microorganisms[(45)].

France Ncube et al., 2017 has conducted a study to assessingbio aerosols sampling, occupational noise, thermal conditions measurement, and field based waste compositional analysis. Results showed highest exposure concentrations for Gram-negative bacteria (6.8×10^3 cfu/m^3) and fungi (12.8×10^3 cfu/m^3), in

the truck cabins. Soincreased risk of exposure for refuse bin loaders and truck cabin samples[49].

Steiner et al., 2005, Subjects (778 wastewater, garbage and control workers; participation 61%) underwent a medical examination, lung function tests [American Thoracic Society (ATS) criteria], and determination of CC16 and SPB (Serum Clara cell protein (CC16) and serum surfactant protein B (SPB) . Inhalation of bio aerosols has been hypothesised to cause "toxic pneumonitis" that should increase lung epithelial permeability at the bronchioalveolar level. Serum Clara cell protein (CC16) and serum surfactant protein B (SPB) have been proposed as sensitive markers of lung epithelial injury. This study was aimed at looking for increased lung epithelial permeability by determining CC16 and SPB in workers exposed to bio aerosols from wastewater or garbage. The increase in CC16 in serum supports the hypothesis that bio aerosols cause subclinical "toxic pneumonitis", even at low exposure[50].

PATHOGENESIS

The mucosa of the lung contains specialized adaptive immune cells (e.g., T lymphocytes with limited antigen recognition abilities and plasma cells that synthesize a non-complement-binding antibody, IgA) and innate immune cells (e.g., alveolar macrophages [AM], natural killer [NK] cells, and dendritic cells). These cells limit the immunological and inflammatory responses to foreign substances that enter the respiratory system.

AMs provide an important link between the alveolar spaces, the "Achilles heel" post terminal bronchiole region, and the mucociliary clearance system.

AMs can also suppress T cell activity by direct contact with the T cell or by the secretion of soluble factors such as nitric oxide, prostaglandin E2, and the immunosuppressive cytokines IL-10 and transforming growth factor β (TGF-β). The ability of the alveolar macrophage to dispose of foreign material rapidly and without mounting an inflammatory response enhances the lung defencesystem.

Kalahasti et al., 2010, The present study assessed the relationship between occupational health hazards and serum pro-inflammatory cytokines in workers engaged in hazardous waste area. The levels of serum pro-inflammatory cytokines IL-8 & TNF- α were significantly associated with subjects who had respiratory symptoms & further supporting that they are inflammatory markers in respiratory symptoms [(35)].

Wouters et al., 2002, have reported the association between nasal lavage concentrations of IL8 and respiratory symptoms in workers exposed to biological hazards (endotoxin, (1→3) -β -Dglucan, viable fungi and it spores and fungal extra cellular polysaccharides) from landfill area. The levels of nasal lavage interleukin-8 considered as upper airways inflammation responses but not on lower airways inflammation responses. Also influenza-like disorders (rhinitis, conjunctivitis, cough, headache are common [(36)].

Anwar SK et al, Arora R et al[51]., 2016, The authors have mentioned that inhalation of dust particles which reach the smaller bronchioles, can eventually activate receptors like c type lectin receptors, protease activated receptors among others, present in the airway epithelial cells, leading to a cascade of immune process, resulting in acute inflammation. They say that the reduction in FEV1 may be due to the loss of elastic recoil pressure of the lungs, which decreases the force required to expel air out of lungs , due to the microscopic enlargement of air spaces but not due to visible emphysema [51–53].

Heldal et al., 2003, In a recent study made in 25 waste handlers at a landfill near Oslo, the Spiro metric FEV1 values were significantly lower at the end of the workweek than at the beginning of the week. Cytological examination of sputum showed an increased percentage of neutrophils and IL-8,supporting the hypothesis that an etiopathogenic mechanism of an a specific inflammatory nature, mediated by neutrophils, underlies the Spiro metric alterations. They have reported higher levels of neutrophils and interleukin-8 in induced sputum of workers exposed to bio-aerosols (endotoxin & (1→3) -β -D-glucan) during waste collection. However, it has been shown that moderate exposure to fungal spores and betaglucans, and even low exposure to endotoxin during waste handling, induces upper airway inflammation characterized by neutrophil influx and activation (myeloperoxidase, eosinophil cationic protein and interleukin-8) .These investigations were characterized as inflammatory responses in the lower airways[54].

Iyawe and Ebomoyi, et al., 2005 in their study showed significant decrease in PEFR in urban solid waste workers may be due to inhalations of gaseous chemicals, obnoxious odours, and deposition of dust and inspirable particulate matter along the respiratory tract. These cause inflammatory changes which leads to increased airway resistance thereby bringing about the remodelling of the airway and consequently lung dysfunction The exposure-dependent decrease in the PEFR observed in the test groups when compared with the control may be due to the direct inhalation of a larger and progressively accumulating volume of obnoxious gaseous chemicals and particulate matter deposits in the lungs with associated inflammatory changes, as well as physically impeding the normal lung function [55–57]. Peak expiratory flow rate (PEFR) is one of the important and simple respiratory function tests, as well as in the early diagnosis of occupational lung diseases [52].

Raveena Ragavi et al., 2016, showed a decrease in FEF 25-75 in sanitary workers, which might be due to airway obstruction (zuskin et al 2016) resulting with accumulation of dust, smoke, bio aerosols and circulation of other mediators which can led to tissue remodelling like hypertrophy in the micro airway of the lung. These sanitary workers are prone to exposure to the environmental pollution. PEFR was signicantly decreased in sanitary workers which might be due to decreased in respiratory muscle strength (Symth et al 1984)

These workers are also presented with abnormal respiratory symptoms. Finally this study also showed that along with the altered PFT parameters and the respiratory endurances parameters will support to the finding shows the sanitary workers of Kanchipuram population are also prone for the small airway obstruction likely because of chronic exposure to environmental pollution, chemicals and other bio-aerosol on a long term basis[58].

EFFECT OF SOCIO- ECONOMIC & EDUCATIONAL STATUS

Jayakrishnan T et al., 2013, Cross-sectional descriptive study showed waste work isoverridden by the social, economic, and environmental deprivations and also involves gender issues. The working conditions for women sweepers are often very poor, they may have no protective wears or equipment's but few complain about the situation.This occupation is physically strenuous, resulting in high pulmonary ventilation and requiring workers breathing through their mouth rather than nose. Studies have shown that relative energetic loads, expressed as oxygen consumption, are significantly higher for waste collectors than recommended limits[59,60].

TYPE AND DURATION OF WORK

Mohammad Shadab et al., 2013 in their study to see the effect of different types of dusts, Bio-aerosols and fume in occupationally exposed person that is Street cleaners (working for more than five years). Cross Sectional Study on 110 Street cleaners (working for more than five years) of which 80 were non-

smokers and 30 were smokers and 60 Control subjects of which 30 were non-smokers and 30 were smokers. Occupational exposure of the workers to harmful dust, gases and bio-aerosols leads to Obstructive type of impairment of Lung functions (working for more than last five years) which is aggravated by smoking [30]=

Srinivasan Roopa et al., 2012, This paper presents the results of pulmonary function assessment in 178 conservancy workers (100 sweepers & 78 loaders) of the Chennai Corporation .Prevalence of respiratory symptoms, low lung function values than a normal healthy person , increasing impairments with increasing years of working were noted probably due to occupational exposures. Sweeping, loading and unloading solid wastes could potentially expose workers to greater than safe levels without protection[53].

Harrisons principal of internal medicine says, inhaled dust and cement particles get lodged in the lung and causing lung irritation, excess mucus secretion in response initially, followed by functional lung impairment thus lung inflammation and chronic obstructive or restrictive lung diseases and pneumoconiosis later[54,61].

Carlos et al., 1999, in their study has said sweeping, vehicle movement turns dust to finer particles thus inhalation reaches lower down the respiratory tract resulting in respiratory problems in sweepers on road [62].

All the studies have used Spirometry to assess lung functions.

Nowadays modern computerized pulmonary function systems allow very sophisticated data handling and storage, graphic display of manoeuvres, accurate calculation and enhanced reporting method. They include physical transducers, analog to digital converters and computer software to process and record the data. Microprocessor based portable spirometers are now available [19].

EFFECT OF POLLUTANTS ON PULMONARY FUNCTION TESTS in SANITARY WORKERS.

INCREASED RESPIRATORY SYMPTOMS

1. **Malta-Vacas et al.,** 2012, in their study showed respiratory symptoms like cough was predominant among all groups of waste collectors increasing their exposure chemical and biological sensitizers in the workplace[47].

2. **Abbasi et al.,** 2012, a cross sectional survey was conducted between January - March 2009 on a sample of 200 adults selected from two villages of district Khairpur, Sindh, Pakistan. A modified version of the American thoracic society division of lung disease questionnaire was used to record the presence of respiratory symptoms. Presence of respiratory symptoms was significantly associated with restrictive and/or obstructive patterns after controlling for confounders[63].

ABNORMALITIES IN PFT PARAMETERS

Fahim D. 2013., in his cross sectional studyon 44 hospital sanitary workers and 57 ministerial workers in General Public Hospital in Ismailia, Egypt., increased respiratory symptoms and decline in some of the pulmonary function parameters .Sanitary workers had more complaints of productive cough (27.2%) as compared to 15.7% of the un-exposed group (p = 0.158). Among sanitary workers 33% reported wheezing and 24% dyspnoea. These symptoms were higher in the exposed group compared to the un-exposed group (p = 0.031 and p=0.011 respectively). Also mean {predicted values} of FEV1/FVC, PEFR,FEF 25 – 75% were lower than the unexposed group. He says further research on work tasks, types of exposures and nature of pulmonary dysfunctions needs to be evaluated[(64)].

Urch et al. (2005), said in their study that exposure to air pollution has been shown to cause arterial vasoconstriction and alter autonomic balance. Responses during 2-hr exposures to concentrated ambient fine particles (PM2.5) plus ozone (CAP+O3) were compared with those of particle-free air (PFA) in 23 normotensive, non-smoking healthy adults. observed an increase in DBP of about 6mmHg in subjects exposed .**Gong et al.,** 2003, in their study reported an increase in the systolic blood pressure in healthy subjects on exposure to particulate matter (PM); and also with the report of Jessup et al (2009) who observed an increase in systolic dysfunction due to exposure to toxins and pharmacological agents which cause intracellular damage and oxidative stress.

Also, some of these inhaled chemicals and gases may possess vasoconstrictor effects, thus increasing the total peripheral resistance and hence increase the blood pressure[55, 66, 67].

M. C Matheson et al., 2005, conducted a study in 1232 general population .They were made to fill a detailed respiratory questionnaire, Spiro metric testing ,gas transfer measurement and Job histories were coded. The prevalence of emphysema,chronic obstructive bronchitis and COPD were in that decreasing order. Subjects ever exposed to biological dusts had an increased risk of chronic obstructive bronchitis, emphysema and COPD. These risks were higher in women than in men. Mineral dust or gases/fumes showed no increased risks for COPD [70].

Neghabet al., 2013, in his study quotes that, there is evidence that exposure to Bio aerosols such as fungi, 1-3 beta-glucan, endotoxin can cause inflammation in the respiratory airways. Similarly, exposure to bio aerosols may increase respiratory symptoms and often causes flu-like symptoms and chronic obstructive pulmonary disease (COPD).In some studies high atmospheric concentration of bio aerosols has been found in the breathing air of workers engaged in disposal of waste, or employed in waste recycling sites, collection and transferring stations of solid wastes, poultry industry, slaughterhouses, hospitals and food processing units[13,38,41,70].

HalimIssever et al.,(71), they correspond well with the Spiro metric pattern of obstructive ventilatory disorders where lung capacity (TLC) total and FVC are either normal or increased, but the hallmark is a decreased expiratory flow rate, usually measured by FEV1. Thus, the ratio of FEV1/FVC is characteristically decreased[(71)].

Cointreau-Levine S., 1998, says this occupation is physically strenuous, resulting in workers breathing through their mouth rather than their nose. Individuals who breathe through their mouth have higher pulmonary ventilation rates when comparing to 10 those who breathe through their nose [(59)].

De Souza et al., 2016,in their study also showed decrement in lung functions probably due to endotoxin and bio aerosol exposure [(72)].

Zuskin et al. reported that the baseline ventilatory capacity was significantly decreased compared with the predicted values in sewage workers. In particular, the values for FEF25 -50 were reduced, [23] suggesting obstructive changes in smaller airways. They mentioned that sewage workers are exposed to different occupational noxious agents, which may lead to the development of chronic lung function changes [(73).]

de Meer G et al.,2007, in their study results have shown exaggeration of pre-existent airway inflammation in organic waste loaders with regular respiratory symptoms.Only a slight reduction in FEV1 was observed in a group of 16 organic waste loaders, only 6 of whom had a positive history for

respiratory symptoms, which was determined by questionnaire. The authors concluded that the occupational exposure could be responsible for exacerbating airway inflammation only in those subjects with pre-existing respiratory symptoms [74,75].

Jahangiri M et al., 2015, 198 employees of wastewater treatment plants and 99 unexposed persons were studied. American thoracic society (ATS) standard respiratory symptom questionnaire was used to determine the prevalence of respiratory symptoms. Pulmonary function tests were conducted for each participant. The prevalence of respiratory symptoms among exposed persons was significantly higher than that of unexposed people. Mean values of most pulmonary function test parameters were significantly lower in the exposed compared to the comparison group persons [76].

Luigi Vimercati et al., 2016, study was done in 124 subjects, 63 waste collectors, and 61 office clerks using Spirometry and questionnaires.FEV1% and FEV1 was reduced in exposed workers, as compared with the controls. FVC no difference noted .Advised PPE usage [74].

Athanasiou et al., 2010, in their study on 104 municipal solidwaste disposal workers and a control group of 80 office workers has shown reduction in FVC, FEV1, FEV1/FVC. They also showed that respiratory symptoms, investigated by means of a validated questionnaire, were also significantly more common in the exposed group than the control group[43].

Ray et al., 2004, in a study conducted in Delhi, India in 96 solid waste disposal workers at an open landfill. A significant reduction in FEV1 and the Tiffenau Index values was found in the exposed workers compared with the controls.Also found statistically significant prevalence of respiratory symptoms in the exposed group.The physical effort and muscle work that these activities entail, during lifting and manhandling of the various containers. This induces a ventilation response consisting of an increased flow volume and respiratory frequency [77,78].

In developing countries like India, there has been little study of the health and injury incidence of solid waste workers. Most of the reviewed studies suffer from limitations related to poor exposure assessment, and lack of information on relevant confounders. In these contexts, to understand and assess the health-risks involved in waste management, this study was conducted among sanitary workers in a tertiary care hospital, where the problem has worsened due to high consumption pattern and per capita solid waste generation and low per capita availability of resources.

AIM & OBJECTIVE

AIM

To evaluate the occupational respiratory dysfunction among sanitary workers.

OBJECTIVES

1. To evaluate the respiratory functions in sanitary workers and normal healthy individuals.
2. To compare the effect of duration of exposure on respiratory functions in sanitary workers.

MATERIALS AND METHOD

STUDY DESIGN - Cross-sectional study

PERIOD OF STUDY - JAN 2018 to DEC 2018

PLACE OF STUDY - Department of Physiology
Chengalpattu Medical College

SUBJECTS

Institutional ethics committee approval was taken. Participants were explained about the details of the study and informed written consent was obtained. 60 hospital workers (Group I) involved in waste collection and disposal of both gender, were selected as cases and were compared with age and gender matched healthy subjects (Group II) not exposed to different waste collecting airborne contaminants. Convenient sampling done. Detailed history and clinical examination was carried out to rule out any acute or chronic illness. Information regarding respiratory illness, frequency and symptoms noted.

INCLUSION CRITERIA	EXCLUSION CRITERIA
• Age group 20-45years • Both gender • Hospital sanitary workers • Work experience > 2 year	• H/O Respiratory disorders /on treatment • Kyphoscoliosis • Smokers • Alcoholics • H/O previous surgeries. • Pregnancy • H/o Diabetes mellitus, Hypertension and any chronic illness. • HIV positive • Recent myocardial infarction • Cardiac disease like unstable angina • Connective tissue disease • Post thoracic surgery • Chest / abdominal pain /oral / facial pain of any cause

Data was collected using interview technique using semi- structured questionnaire.

1. General information – name, age, gender, address.
2. Socio- demographic features – education, occupation, family type, educational qualification & occupation type, socio-economic status
3. Acute morbidity: any morbidity in the past 3 months (both reported and at present) included questions on respiratory (presence or absence of regular dry and/or productive cough, phlegm, wheezing, shortness of breath, etc.),

nasal and eye symptoms, smoking habits, medical and family history of each subject

4. Chronic morbidities (both reported and at present).
5. Awareness, availability and practice regarding personal protective equipment.
6. Treatment seeking behaviour.
7. Working hours
8. Years of service
9. Anthropometric measurements.

   Height in cms.

   Weight in kgs.

   Body mass index (BMI) was measured using the formula Weight (in kg)/height (in m^2).
10. Physical examination and investigations.

Pulmonary Function Test examination was done to assess the respiratory health.

PFT was recorded using Spirometer **Easy On PC Spirometry** (nddmedizintechnik AG, Zurich, Switzerland).

The apparatus uses ultrasound flow sensor. It measures the transit time which allows the accurate determination of flow velocity independent of temperature, humidity and molar mass of the gas. Measurement is digitalised .So the sensor requires only one single calibration and does not change during the sensor's lifetime.

METHODOLOGY

The pulmonary function parameters;

1. Forced Vital Capacity [FVC],
2. Forced Expiratory Volume in 1 second [FEV1]
3. Forced Expiratory Flow [FEF25–75]
4. Peak Expiratory Flow [PEF] were recorded according to the American Thoracic Society criteria.

ACCEPTABILITY CRITERIA

According to American Thoracic Society Criteria

- Effort should be maximal, smooth and cough free
- Exhalation time should be for a minimum of 6 seconds
- End of test is indicated by 2-second volume plateau [22,24]

REPRODUCIBILITY CRITERIA

According to American Thoracic Society Criteria

- Two largest FVC measurements should be within 200ml of each other
- Two largest FEV1 measurements should be within 200ml of each other[22,24].

ON THE DAY OF RECORDING

Subject was advised to avoid

1) Eating meals 2 hours prior to the test
2) Drinking alcohol 4 hours prior to the test.

3) Using short acting bronchodilators 6 hours prior to the test

4) Using long acting bronchodilators 12 hours prior to the test

SPIROGRAM (OPEN CIRCUIT METHOD)

- Subject was seated comfortably; sit straight.
- Demonstrate how to do the procedure
- Subject was asked to inhale air deeply to fill the lungs
- Nose clip is put
- Spirette is placed into mouth with lips tightly sealing around it.
- Subject is asked to blow out as fast as possible at least for 6 seconds.
- Minimum of 3 trials done at an interval of 5 minutes
- Best of three trials are taken for analysis [19].

ANALYSIS OF PFT VALUES

Percentage of the Predicted values of

FEV1

FVC

FEV1 / FVC

FEF25% -75%

PEFR were taken for analysis. (18)

The values (FVC, FEV1 and FEV1/FVC) were compared with average predicted for a subject on the basis of age, sex, built and race.

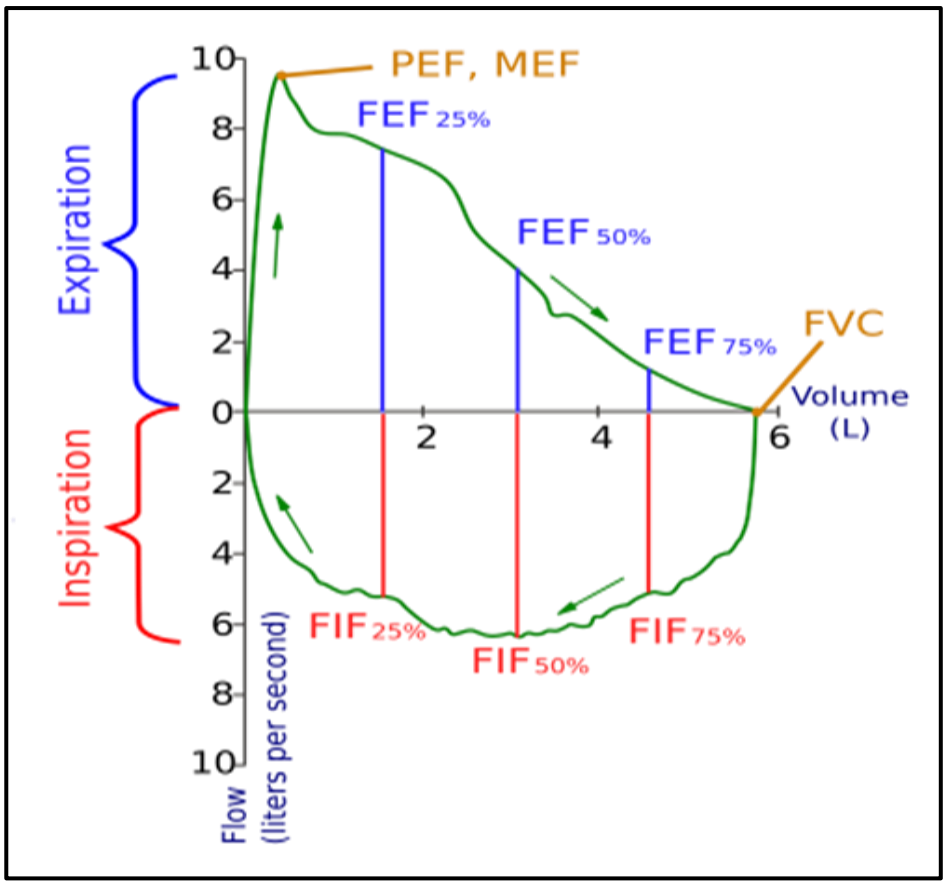


Figure12: Flow – volume loop

Table 5: Patterns of Abnormalities in Pulmonary Function Test Results

Pulmonary Function Measurement	Obstructive Pulmonary Disease	Restrictive Pulmonary Disease
FVC (L)	Decrease	Decrease
FEV_1 (L)	Decrease	Decrease
FEV_1/FVC	Decrease	Normal
FEF_{25-75} (L/sec)	Decrease	Normal to increased
PEFR (L/sec)	Decrease	Normal
FEF_{50} (L/sec)	Decrease	Normal
FEF_{75} (L/sec)	Decrease	Normal
Slope of FV curve	Decrease	Normal to increased

ANALYSIS OF DATA OBTAINED

The parameters were analysed by SPSS version 16.0. Descriptive statistics was made for various parameters. The mean and standard deviation for the data was calculated. The values of pulmonary function parameters of sanitary workers were compared with controls and their significance was obtained using Independent sample‘t’ test. The pulmonary function parameters were compared with the duration of work among subjects by using Pearson’s correlation coefficient test.

RESULTS

A cross sectional descriptive study was conducted among sanitary workers in a tertiary care centre. Totally 120 participants took part in the study. Out of 120 participants 60 formed the study group (7 males and 53 females) who were the hospital sanitary workers and the remaining 60 (6 males and 54 females) were normal subjects not exposed to medical wastes formed the control group. The study groups were divided into 3 subgroups based on work experience.

Group I - work experience <3 years

Group II - work experience 3 -5 years

Group III - work experience >5 years, based on the duration of employment in sanitation handling wastes.

The workers often had acute respiratory illness, tiredness, followed by gastroenteritis occasionally and also injuries for which they sought treatment as OP. Most of the workers being ladies were clinically anaemic.

Table 6 : Descriptive statistics of anthropometric parameters

Anthropometric Parameters	Group	N	Mean	SD	P
Age	Cases	60	40.4	5.582	0.426
	Controls	60	39.58	5.613	
Height in cm	Cases	60	153.33	7.776	0.523
	Controls	60	154.05	3.811	
Weight in Kg	Cases	60	54.12	9.743	0.072
	Controls	60	56.98	7.366	
BMI Kg/m2	Cases	60	23.704	3.8464	0.701
	Controls	60	23.946	2.9779	
	Controls	60	81.92	9.011	

This table shows the anthropometric data of subjects and control groups. The mean and standard deviation of anthropometric measurements of subjects and control groups were calculated. The results were not statistically significant.

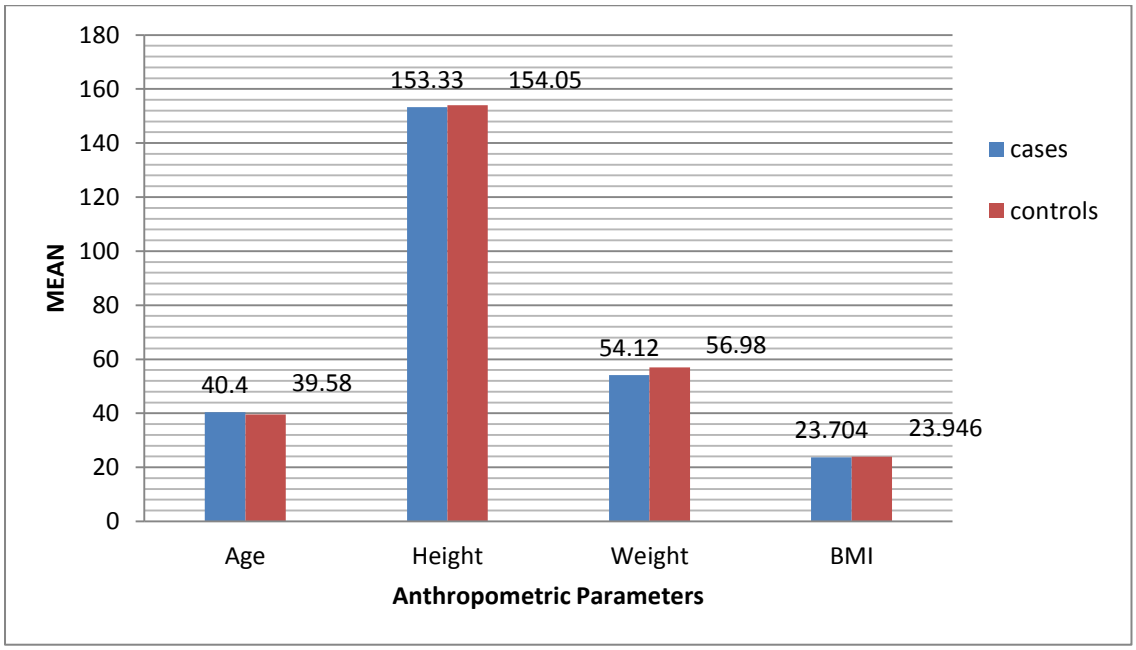


Figure 13 : Anthropometric parameter distribution of controls and Subjects.

This figure shows subjects and controls are comparable.

Table 7: Table showing Exposure in Years amongSubjects

GROUP	YEARS	No of Workers
I	<3	21
II	3 TO 5	18
III	>5	21
	Total	60

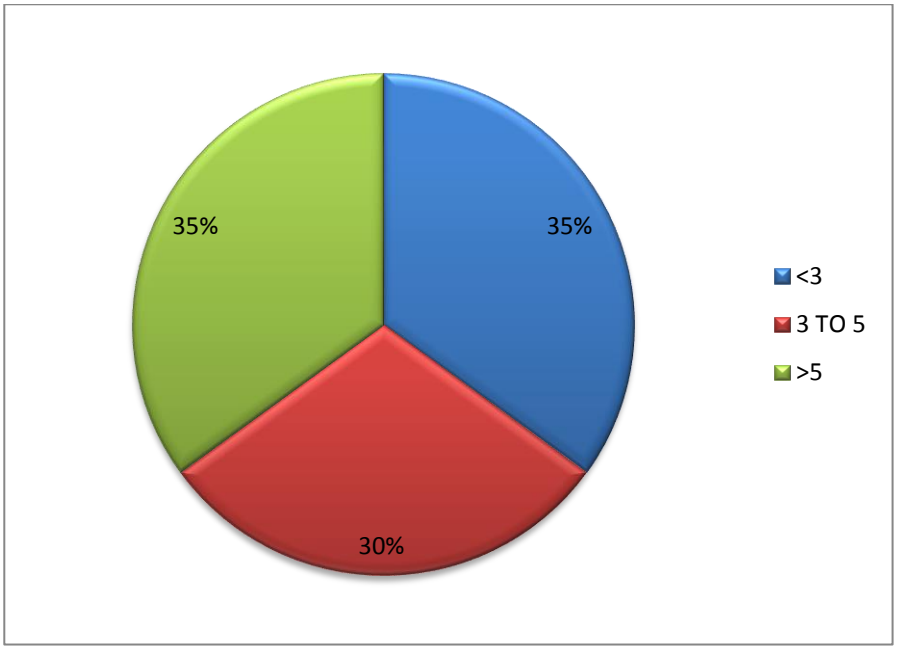


Figure 14 : Pie chart showing work experience among subjects

This pie chart shows distribution of sanitary workers based on their years of employment and exposure to dust etc., on average they had completed 3-5 years of service.

Table 8 : PFT values of controls and subjects

Pulmonary Function Parameters	Group	N	Mean	SD	P
FVC	Cases	60	62.8	9.896	**0.0001**
	Controls	60	92.62	14.958	
FEV1	Cases	60	62.07	11.087	**0.0001**
	Controls	60	93.7	15.915	
FEV1/FVC	Cases	60	97.97	9.13	0.064
	Controls	60	100.5	5.15	
FEF25-75%	Cases	60	71.5	18.3	**0.002**
	Controls	60	79.92	8.939	
PEF	Cases	60	75.17	19.976	**0.02**
	Controls	60	81.92	9.011	

(P< 0.05 is considered statistically significant)

This table shows the pulmonary function parameters of subjects and control groups. Statistically significant reduction in FVC%, FEV1, and FEF 25-75% values. PEF was also reduced but not significantly.

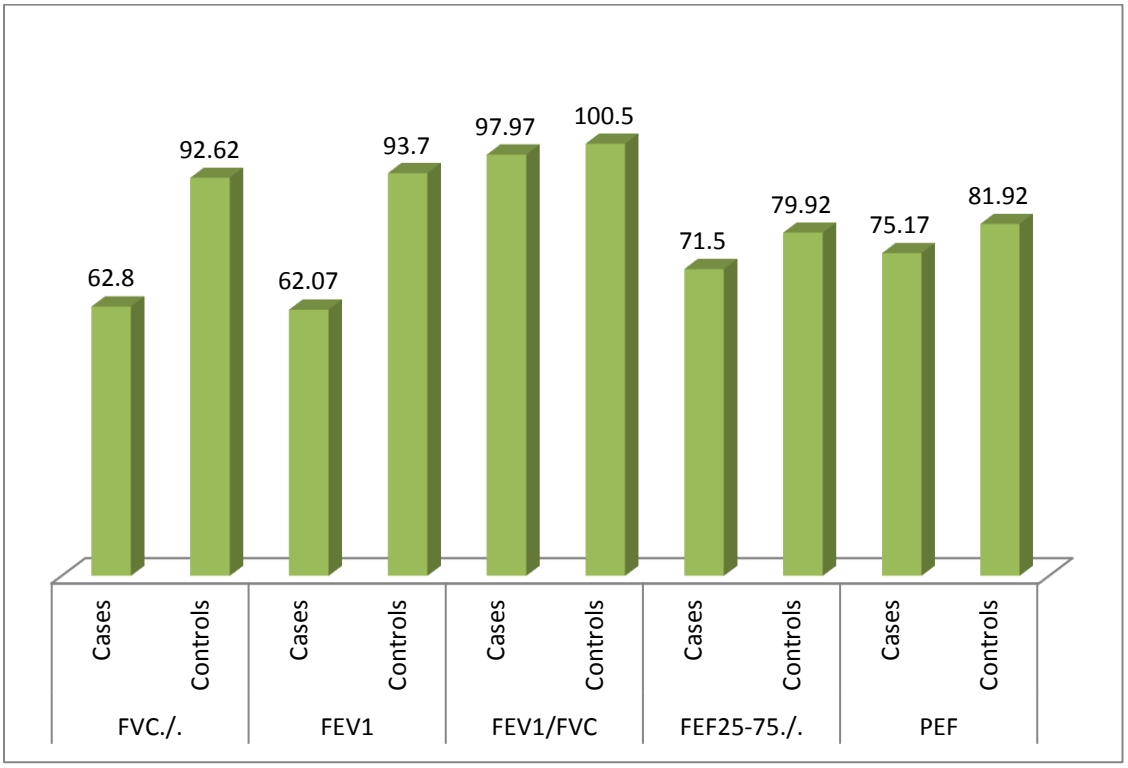


Figure 15 : Bar chart showing pulmonary function parameters

It is observed from this figure that, FVC%, FEV1, FEF 25-75%, PEF are reduced in subjects compared to cases.

Table 9 : Pattern of Spiro metric Report in controls and Subjects.

Pattern	Cases	Controls	Total	Chi sq	P
Normal	0	50	50	87.88	**0.001**
Restrictive	58	8	66		
Obstructive	2	2	4		
Total	60	60	120		

This table shows majority of workers had restrictive type of Spiro metric recording when compared to normal recording in controls.

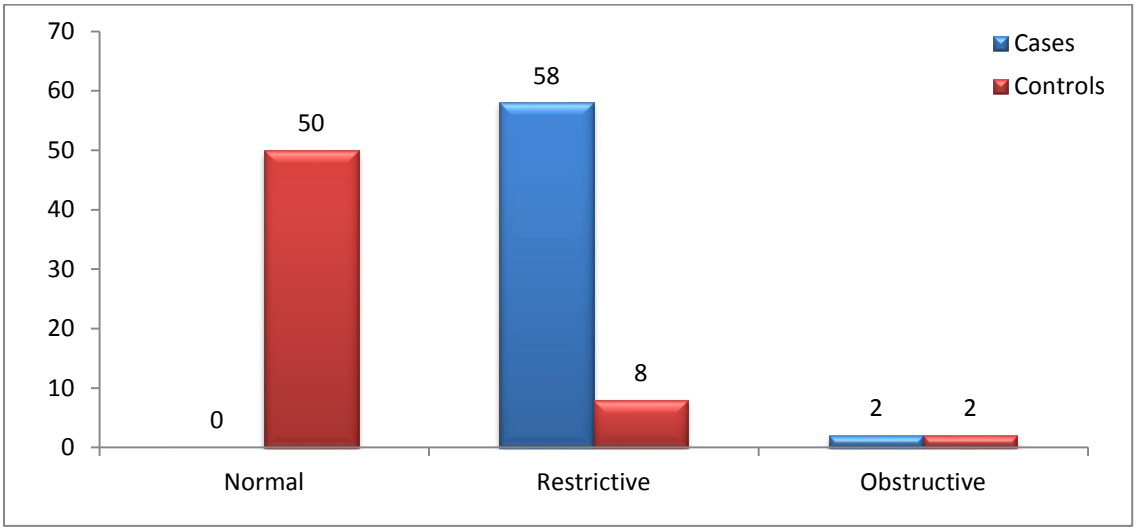


Figure 16: Bar Chart showing distributionpattern of spirometry values in controls and Subjects.

It is observed from this figure that, sanitary workers had a restrictive type of recording on spirometry.

Table 10: Comparison table of FVC% among subjects and controls

Parameter	Group	N	Mean	SD	t	P
FVC./.	Cases	60	62.8	9.896	12.878	**0.0001**
	Controls	60	92.62	14.958		

P< 0.05 is taken as statistically significant value. Here P is 0.0001 highly significant meaning FVC % is reduced among workers.

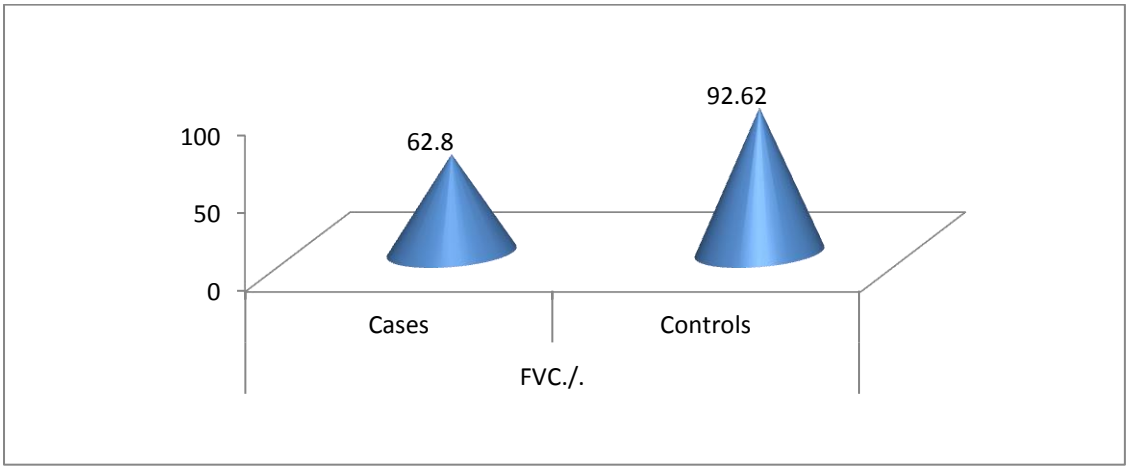


Figure 17: Bar chart showing comparison of FVC% among subjects and controls

The bar chart clearly shows the reduction in FVC % in workers when compared to normal values in controls.

Table 11 : Comparison table of FEV1 among subjects and controls

Parameter	Group	N	Mean	SD	t	P
FEV1	Cases	60	62.07	11.087	12.633	**0.0001**
	Controls	60	93.7	15.915		

P< 0.05 is taken statistically significant value.

Here we can see FEV1 is reduced among workers compared to controls and reduction is statistically significant with a P value of 0.0001. Both FVC and FEV1 are reduced among workers.

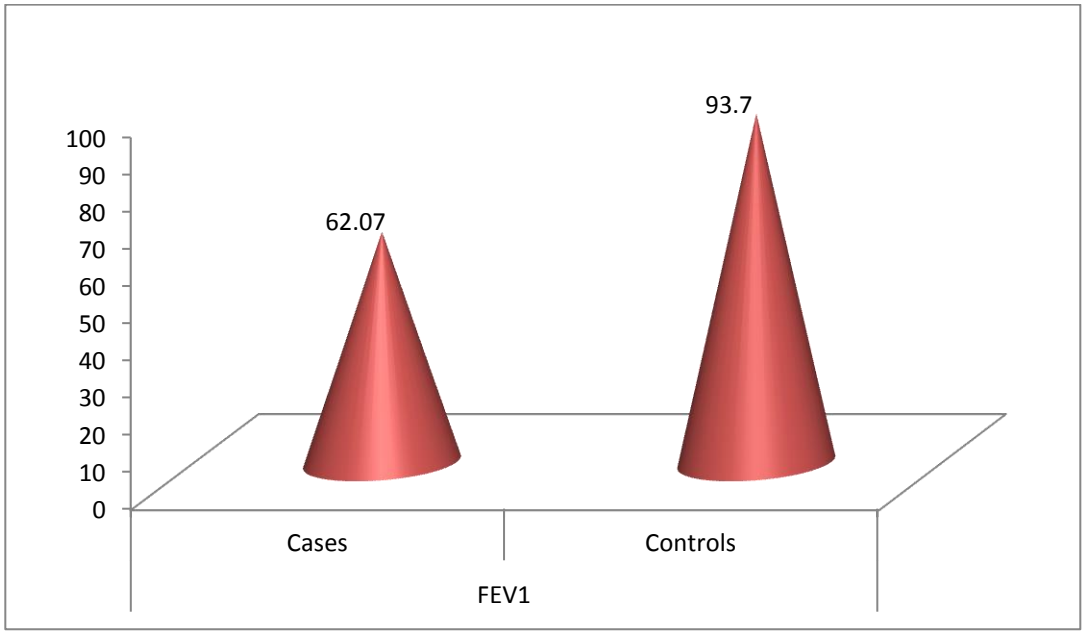


Figure 18: Bar chart showing comparison of FEV1 among subjects and controls

The bar chart clearly shows the reduction in FEV1 in workers when compared to normal values in controls.

Table 12 : Comparison table of FEV1/FVC% among subjects and controls

Parameter	Group	N	Mean	SD	t	P
FEV1/FVC	Cases	60	97.97	9.13	1.872	0.064
	Controls	60	100.5	5.15		

(P< 0.05 is taken statistically significant value)

This table doesn't show statistically significant difference in FEV1/FVC values among subjects and controls.

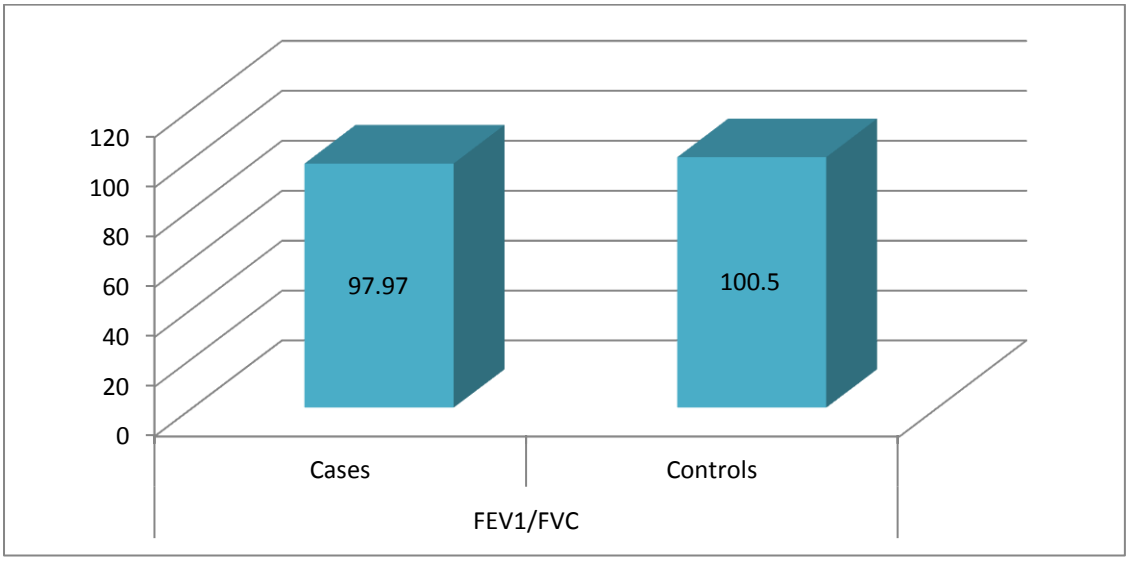


Figure 19: Bar chart showing comparison of FEV1/FVC% among subjects and controls

The bar chart clearly shows the reduction in FEV1/ FVC % in workers when compared to normal values in controls but not statistically significant.

Table 13 : Comparison table of FEF25-75./ among subjects and controls

Parameter	Group	N	Mean	SD	t	P
FEF25-75./.	Cases	60	71.5	18.3	3.091	**0.002**
	Controls	60	79.92	8.939		

(P < 0.05 is taken statistically significant value)

Here FEF25-75% is reduced in workers compared to controls with a P value of 0.002 statistically highly significant.

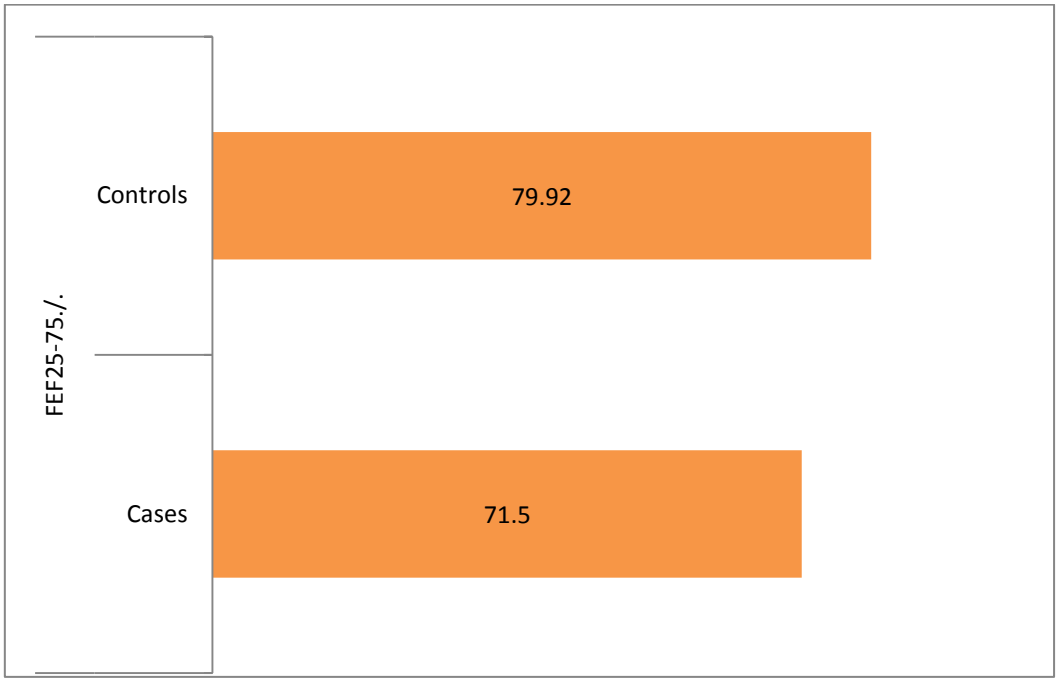


Figure 20: Bar chart showing comparison of FEF25-75% among subjects and controls

The bar chart clearly shows the reduction in FEF25-75 % in workers when compared to normal values in controls .

Table 14: Comparison table of PEF% among subjects and controls

Parameter	Group	N	Mean	SD	t	P
PEF	Cases	60	75.17	19.976	2.386	**0.02**
	Controls	60	81.92	9.011		

(P< 0.05 is considered statistically significant.)

This table doesn't show statistically significant difference in PEF values among subjects and controls.

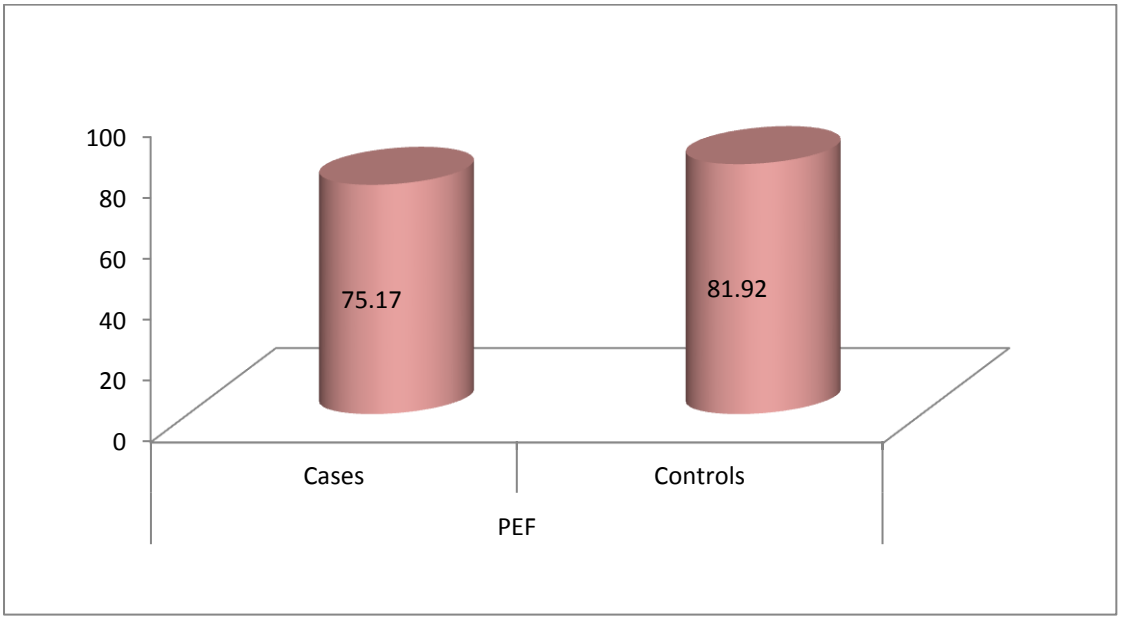


Figure 21: Bar chart showing comparison of PEF% among subjects and controls

From above data it is evident that all PFT parameters are better for controls compared to subjects statistically significant except FEV1/EVC ratio.

Table 15: Comparison table of FVC% among subjects with duration of exposure

Years of Exposure	<3Yrs	3-5Yrs	>5Yrs
FVC%	71	64	54

This table shows decrease in FVC % values with increasing years of employment in sanitary workers.

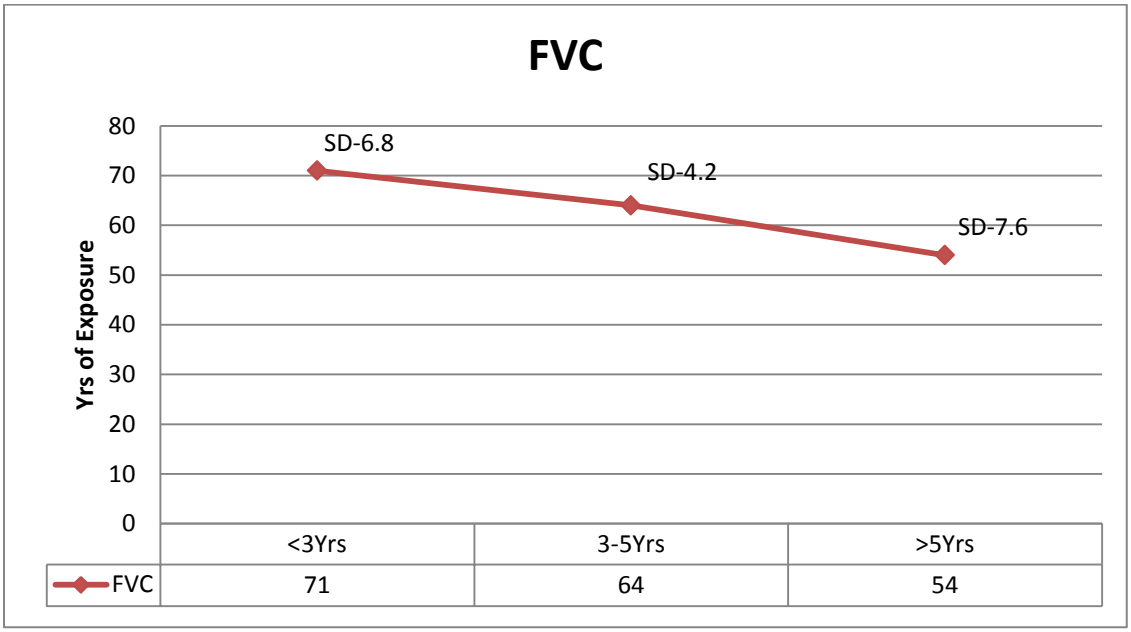


Figure 22: Line Diagram showing comparison of FVC% with duration of exposure

The line diagram clearly depicts the declining trend in FVC % with increasing duration of employment.

Table 16: Comparison table of FEV1 among subjects with duration of exposure

Years of Exposure	<3Yrs	3-5Yrs	>5Yrs
FEV1	71	63	53

This table shows decrease in FEV1 values with increasing years of employment.

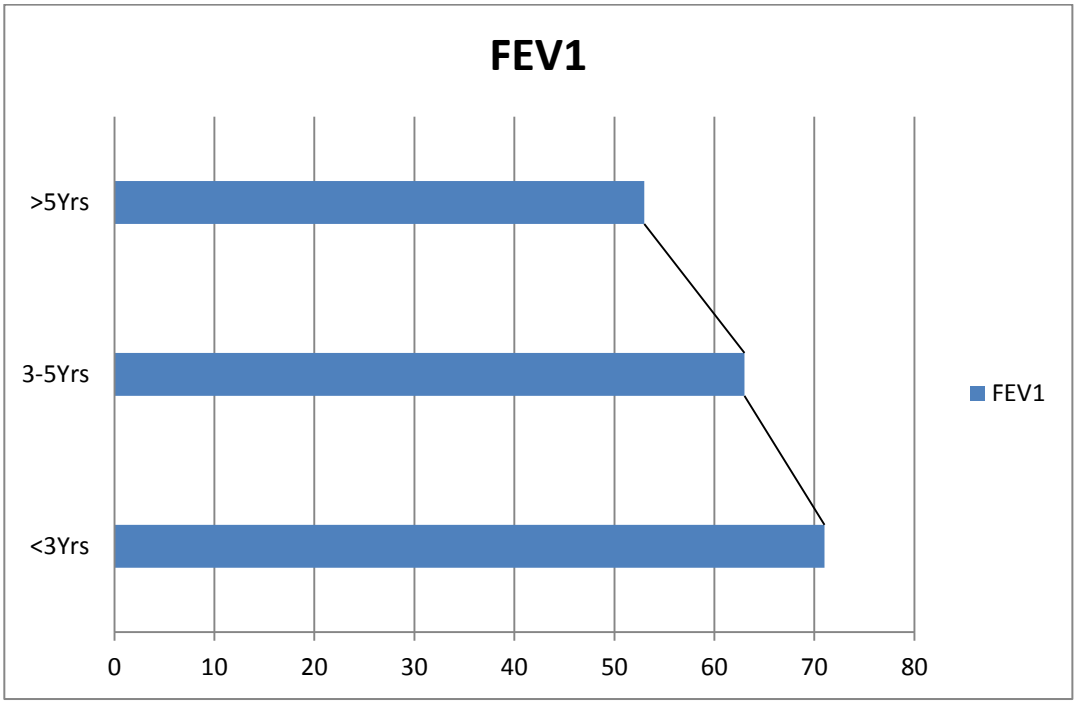


Figure 23 : Bar chart showing comparison of FEV1 with duration of exposure

The bar diagram clearly depicts the declining trend in FEV1 with increasing duration of employment.

Table17: Comparison table of FEV1/FVC% among subjects with duration of exposure

Years of Exposure	<3Yrs	3-5Yrs	>5Yrs
FEV1/FVC	100	96	98

This table shows no significant difference being a ratio between FEV1 and FVC values.

Table 18: Comparison table of FEF25-75%among subjects with duration of exposure

Years of Exposure	<3Yrs	3-5Yrs	>5Yrs
FEF25-75	76	67	71

This table shows reduction in values but not showing negative trend with years of employment.

Table 19 : Comparison table of PEF%among subjects with duration of exposure

Years of Exposure	<3Yrs	3-5Yrs	>5Yrs
PEF	80	78	68

This table shows decrease in PEF% values with increasing years of employment.

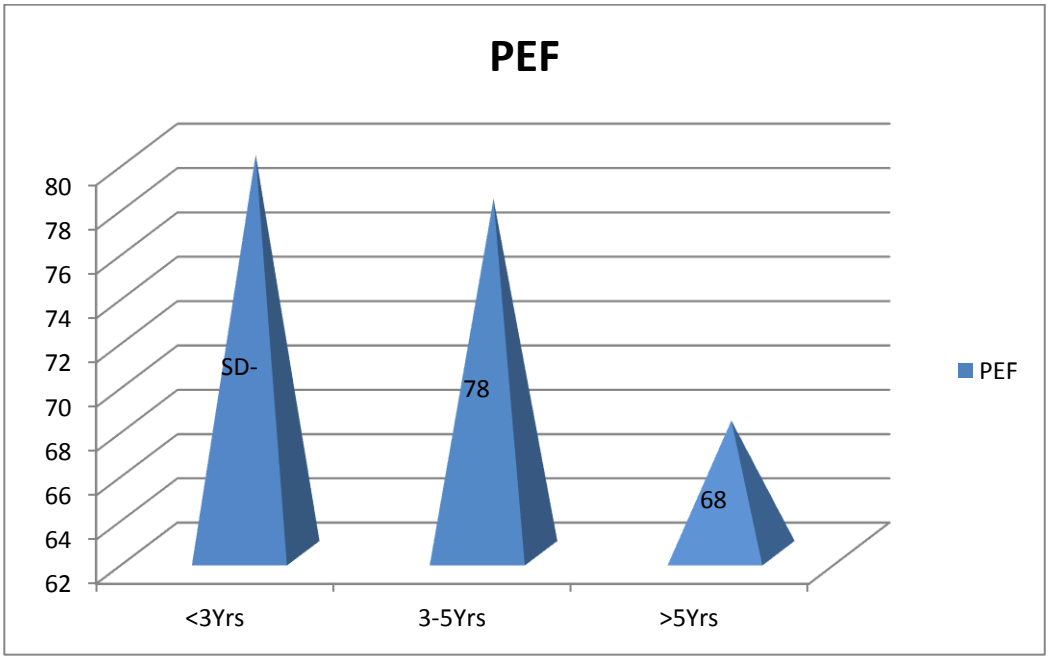


Figure 24: Bar chart showing comparison of PEF with duration of exposure

This bar chart shows decrease in PEF% values with increasing years of employment.

Table 20 : Correlation between FVC of subjects and duration of employment.

Correlation between FVC and duration of employment			
		FVC	**Duration of Employment**
FVC	Pearson Correlation	1	-0.738
	Sig. (2-tailed)		0.001
	Total	60	60
Duration of Employment	Pearson Correlation	-0.738	1
	Sig. (2-tailed)	0.001	
	Total	60	60
**. Correlation is significant at the 0.01 level (2-tailed).			

This table shows statistically significant negativecorrelation between FVC of subjects and their duration of employment. . (R sq linear-0.499), i.e. as the duration of exposure to dust etc., increases with years of service FVC of subjects is found to be decreasing.

Table 21: between FVC of subjects and duration of employment.

Correlation between FEV1 and duration of employment			
		FEV1	**Duration of employment**
FEV1	Pearson Correlation	1	-0.649
	Sig. (2-tailed)		0.001
	Total	60	60
Duration of employment	Pearson Correlation	-0.649	1
	Sig. (2-tailed)	0.001	
	Total	60	60
**. Correlation is significant at the 0.01 level (2-tailed).			

Again this table shows statistically significant negative correlation between FEV1 of subjects and their duration of employment. (R sq linear-0.377), i.e. as the duration of exposure to dust etc., increases with years of service FEV1 of subjects is found to be decreasing.

There was not significant correlation between FEV1/FVC%, FEF25-75% and PEF%.

To summarise: FVC%, FEV1, FEF25- 75%, PEFR are reduced with increasing years of employment in sanitary workers as against normal values in controls.

DISCUSSION

Exposures to dust, bio-aerosols, toxic fumes, gases and various pollutants in the occupational environment is unavoidable in a hospital for a sanitary worker. This is associated with a wide range of health effects, including infectious diseases, acute toxic effects, allergies and cancer. Chemicals used in housekeeping are enlisted as R1 to R9**,** Chemical composition not displayed as per law. Natural resources defence council says air fresheners affects hormones and reproductive health. Toilet bowel cleaners when exposed to skin might cause eye and skin burns. It is dangerous when mixed with other type of cleaners. Cleaning solutions are claimed to be biggest offenders.

Respiratory symptoms and lung function impairment are the most commonly affected, however, valid incidence or prevalence data for most diseases caused by biological agents are lacking.

In our cross sectional descriptive study conducted among 60 sanitary workers in a tertiary care centre with a control of 60 normal subjects not exposed to medical wastes above results were found.

Figure 12 shows control and study group are comparable in their anthropometric parameters such as age, height ,weight & BMI..

The mean age of workers being 40.4±5.58 with 70 % (85) belonging to 35 – 45 age group. Most of the sanitary workers were employed on contractual basis (90 had an experience less than 7 years maximum working for 8 hours

/day. The family size of the workers was 5-6 members, 2 being earning members. Most of the workers had an educational status of 8 to 10th.Those who said 5 th standard could read write. Few were uneducated but can write their names. Socio economic class IV according to Modified Kuppuswamy scale[79] living in nearby villages around the hospital.No major chronic illness noted. An informal introduction on type of work has been explained to them after employment. Gloves and masks have been provided. Knowledge and awareness of using these was there. However regular usage was not practised out of neglect and lack of realisation of risks involved.As most of them were ladies were, involved in sweeping, moping, cleaning toilets in wards, consultation rooms and op departments. They were also involved in works like carrying drugs, saline bottles, pushing stretchers with patients in OP and in shifting patients in OT, also in clearing weeds.

In our study various lung parameters FVC, FEV1, FEV1/FVC, FEF25-75%,PEF were done using Easy on PC spirometer, among sanitary workers of age 25-45 years who were non-smokers and compared with the normal age and gender matched controls not exposed to dust and bio aerosols.

The PFT parameters were compared with respect to the duration of employment in sanitary workers and grouped as <3 years, 3 -5 years, >5 years of work experience. Most of them were contract based workers and workers fitting into inclusion criteria with increasing years of exposure were not available.

Various studies reveals that waste management is a livelihood of people of low educational levels, with insufficient family income, poor living conditions and mostly performed by male employees in India and also other countries[7,60].

Relevance of Questionnaire in evaluation of Frequency of Respiratory Symptoms

From the data collected it is evident that workers have frequent respiratory infections but not hospitalised often.Chronic respiratory symptoms were significantly with decrements in lung function and should be strongly emphasised in clinical or occupational history for assessment of respiratory health. The use of standardised respiratory questionnaires is an effective tool for assessing the burden of respiratory symptoms in occupational setting (80,81)This is similar to various studies mentioned earlier .Hence self-reporting questionnaires to all workers and periodical lung function assays can be used for screening.

Muco-ciliary Clearance System- protects the lower respiratory system by trapping and removing inhaled pathogenic viruses and bacteria, in addition to nontoxic and toxic particulates (e.g., pollen, ash, mineral dust, mold spores, and organic particles), from the lungs . Normally bio aerosols don’t reach alveoli. It is cleared by ciliary action. But particles < 2 micro meter reaches the alveoli. Particles< 0.1 micrometre produce inflammation in the lungs by releasing inflammatory mediators and reactive oxygen species and affects lung

functions.The region from the terminal bronchioles to the alveoli is devoid of ciliated cells and is considered the "Achilles heel" in what is otherwise a highly effective system.

The relatively slow rate of particle clearance in this area renders the terminal respiratory unit the most common location of airway damage for all types of occupational lung disease[14].

Similar pathogenesis has been mentioned in various studies as a cause of lung damage.**Chestnut LG et al.,** 1991, has shown the relationship between pulmonary function and quarterly average levels of total suspended particulates (TSP) indicatingdecrement in FEV1 and FVC [33].

Another study by **Agarwal S et al.,** 2016, has said, size distribution analysis shows that bacteria were mostly abundant in fine particle sizes, i.e. <0.43-2.1 μmbelonging to Bacillus, Staphylococcus, Streptococcus, Klebseilla and Escherichia genera [29].

Similarly in a study by Godleski.J.,et al., 2000, has shown when respirable dust (<10 μm) enters the respiratory system, the human body fights against it as foreign . Increased morbidity and mortality from respiratory and cardiovascular diseases seen with PM exposure [82].

Similarly **Sean H Ling et al**., 2009 study showed that inhalable particulate matter (PM10) shows a strong association with adverse respiratory

health effects, even when adjusted for other major risk factors such as cigarette smoking [17].

Another study by **J. Douwes et al**., 2002, and **Heldal et al.,** 2003, In their review articles says exposures to biological agents in both the occupational and residential indoor environment are associated with a wide range of adverse health effects .Workers in this industry (e.g. waste sorting, organic waste collection and composting) are often exposed to very high levels of microorganisms [34–36].

FVC, FEV1

Figure 14 shows reduction of percentage predicted values of FVC and FEV1 among subjects when compared to the control group. The impairment is more of restrictive pattern. as FEV1 % is normal .

Reviewing literature **Cointreau-Levine S.,** 1998, says that this occupation is physically strenuous, resulting in workers breathing through their mouth rather than their nose. Individuals who breathe through their mouth have higher pulmonary ventilation rates when comparing to those who breathe through their nose[59].

Similarly in a study by Jayakrishnan **T et al.,** 2019, was conducted among solid waste management workers of Kerala, India.They say, this occupation is physically strenuous, resulting in high pulmonary ventilation and requiring workers breathing through their mouth rather than nose. Studies have shown that

relative energetic loads, expressed as oxygen consumption, are significantly higher for waste collectors than recommended limits [59,60].

FEV1%

In our study, the FEV1/FVC ratio is almost normal and did not show any statistically significant difference among sanitary workers compared with controls.

This shows that the sanitary workers had a restrictive type of pulmonary impairment as evidenced by significant reduction in FEV1, FVC and near normal FEV1/FVC ratio. FEV1% signifies obstructive pattern of disease.

Multiple studies as quoted below shows reduction in various PFT parameters with obstructive or a mixed pattern.

Sangolli et al, Luigi Vimerceti and Ray et al., 2004, in a study conducted in Delhi, India in 96 solid waste disposal workers at an open landfill. A significant reduction in FEV1 and the Tiffenau Index values was found in the exposed workers compared with the controls[77].

Ramaswamy et al.,2007 found significant decrease in pulmonary function parameters PEFR, FVC, FEV1 in Sweepers as compared to control subjects. They also reported more decline in Pulmonary Function Test parameters with increasing duration of work in 62% of the Landfill workers employed in disposal of solid waste compared to 27% of the control subjects[77,83].

Mohammad Shadab et al., 2013 in their study on 110 Street cleaners (working for more than five years) of which 80 were non-smokers and 30 were smokers and 60 Control subjects of which 30 were non-smokers and 30 were smokers has shown statistically significant decrease in all PFT parameters. So they found that the Occupational exposure of the workers to harmful dust, gases and Bio-aerosols leads to Obstructive type of impairment of Lung functions(also **HalimIssever et al.,**) **and** (working for more than last five years) which is aggravated by smoking[30].

Neghab et al., 2013, in his study quotes that, exposure to bio aerosols may increase respiratory symptoms and often causes flu-like symptoms and chronic obstructive pulmonary disease (COPD)[13,38,41,70].

Mariammal et al., 2012 , in a study on 101 construction workers and 56 sanitary workers compared with 92 controls showed increased frequency of respiratory complaints .Abnormal Spiro metric findings were significantly higher in the study group, showing that occupational exposure caused harm to the workers respiratory system resulting in the obstructive pattern of lung function impairment.Fahim D et al.,2013, Athanasiou et al.,2010, Jahangiri et al., 2015, show obstructive pattern in their studies.

Though most of the studies shows obstructive pattern of lung impairment our study shows near normal FEV1/FVC%, a restrictive pattern.This may be due to continuous exposure of dust laden with bio aerosols, germs, fungi moulds,

chemical fumes, decompost gases, which in turn causes mucosal oedema affects their respiratory functions.

One more factor for restrictive pattern, poor endurance and effort is that these female workers were anaemic, malnourished leading to muscle weakness.

However though studies showed obstructive type of pulmonary dysfunction, if the condition continued the gradual deterioration of the lung function of the subjects lead to chronic disorders and various complications. The restrictive lung disease resulted in decrease in vital capacity and pulmonary parameters depending upon the duration of exposure to the dust[(84)].

FEF 25-75, PEF

Our study shows decreased values of FEF 25-75 %; PEF though statistically not significant may be due to small airway obstruction arising out of interstitial oedema**.** Smallairways are the major site of damage in persons exposed to dusty environment because relatively small particle size settle their and also escape into alveoli, inducing inflammatory responses. This finally scars the alveoli or causes interstitial oedema.

Moreover they remain in the air for the longer period of time and they also get deposited in lungs in greater amounts than larger sized particles which are cleared in upper airways itself.

Iyawe and Ebomoyi et al.,2005, and RaveenaRagavi et al., 2016, showed a decrease in FEF 25-75 in sanitary workers, which might be due to airway obstruction (zuskin et al 2016) resulting with accumulation of inanimately dust ,smoke, bio aerosols and circulation of other inanimately mediators which can led to tissue remodelling like hypertrophy in the micro airway of the lung. PEFR was significantly decreased in sanitary workers which might be due to decreased in respiratory muscle strength (Symth et al 1984)[58].

Zuskin et al. reported reduced ventilatory capacity in sewage workers. FEF25 -50 were reduced, [23] suggesting obstructive changes in smaller airways. Exposure to occupational noxious agents, may lead to the development of chronic lung impairment[73].

CHANGES WITH DURATION OF EMPLOYMENT

Table 20 and 21 showscorrelation between the duration of employment and FVC, FEV1 respectively. Both PFT parameters are found to decrease with increasing years of employment as seen with studies by **Olayinka Stephen Ilesanmi et al.,** 2014 and **Fahim.D.**

The limiting factor is unavailability of workers with experience >10 years.

The anthropometric influence on declining FVC and FEV1 values is ruled out because only percentage predicted values of subjects are considered.

SUMMARY

- Sanitary workers working in hospital environment are exposed to bacteria laden bio aerosols. This affects all systems especially respiratory system as it is open to external environment.
- Hence this study was done to assess impairment in lung functions.
- The study was conducted in 60 sanitary workers with work experience more than 2 years and 60 age matched healthy normal individuals.
- Spirometry was used to assess lung functions. After IEC clearance, Informed consent taken and test was proceeded.
- Percentage of predicted values of FVC, FEV1, FEV1/FVC AND FEF 25-75% were recorded for subjects and controls.
- A restrictive pattern of recording was noted in sanitary workers compared to normal values in controls.
- This showed that sanitary workers are at higher risk of lung function impairment due to their working environment. Hence protective measures need to be considered.

LIMITATIONS

- One drawback is small sample size of subjects. Hence results could not be generalised to represent whole population.
- Although not very significant statistically other than FVC and FEV1 there is definite decline in lung function test reports. Less significance could have been due to small sample size and wide range of work experience was not noted. Also it was a cross-sectional study would have been influenced by current respiratory status.
- Another limitation of this study is that a temporal relationship cannot be determined due to the cross-sectional design of our study.
- A further limitation is that data related to specific bio aerosols or chemical exposures were not available in this study.

Further studies using a larger sample size and follow up after execution of preventive measures can be considered.

CONCLUSION

Spiro metric analysis of lung functions of 60 sanitary workers , mostly ladies involved in housekeeping work in a tertiary care hospital for >2 years were compared with age matched 60 healthy controls.

- The sanitary workers had reduced lung functions witnessed by decreasing FVC, FEV1 and near normal FEV1/FVC with duration of employment, when compared with control groups .
- In our study,restrictive pattern of lung impairment was noted.
- A decrement in lung functions was noted with increasing years of employment.
- Sanitary workers can be encouraged to use personnel protective equipment PPE.
- Assessment of Lung functions, having a formal session on working environment, type of work, importance of adherence to PPE during job recruitment can be made compulsory.
- Regular monitoring for following PPE can be encouraged.
- Periodic check-ups, preventive vaccination
- Usage of licensed cleaning products will further brighten their future.

- Also reporting of injuries, abstaining from work during illness or scheduling them to less harmful places can be promoted.
- Also in summer work timings may be adjusted to avoid heat exhaustion. Refreshments and cool place to rest and dine can be provided.
- Emphasis on personal hygiene, good nutrition, yoga practices can also be given.

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ANNEXURE

DATA COLLECTION FORM

Name :

Socio economic status :

Age :

Educational status :

Sex :

Occupation :

Years of work :

Working hours :

Type of work :

Present complaints with duration :

Past history :

□ H/o drug intake

□ H/O cardiovascular disease

□ H/O any respiratory illness

□ H/O lung surgery

□ H/O connective tissue disease

□ H/O diabetes mellitus / hypertension

Awareness and practise of of PPE usage :

Knowledge on risk exposure in working environment :

Whether screening, vaccination done during/prior to appointment :

Occupational history :

Family History : H/o of any respiratory illness in family

On examination

Height Vital signs

Weight

General examination

Examination of respiratory and cardiovascular system

Investigations

Spirometry

PATIENT CONSENT FORM

STUDY DETAILS :

"Spirometric Evaluation Of Occupational Respiratory Dysfunction Among Hospital Sanitary Workers In A Tertiary Care Centre "

STUDY CENTRE: Department of Physiology, Chengalpattu Medical College, Chengalpattu.

INDIVIDUAL NAME: AGE: SEX:

IDENTIFICATION NUMBER:

I confirm that have understood the purpose of procedure for the above study.

I have the opportunity to ask question and all my questions and doubts have been answered to my satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw anytime without giving any reasons, without my legal rights being affected.

I understand that my investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study, I understand that my identity

will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arrives from the study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team.

I hereby give consent to participate in this study.

I hereby give permission to undergo complete clinical examination and diagnostic test.

Signature of investigator **Signature / Thumb impression of Participant**

Date :

Place : **Participant's Address**

சுய ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு : மருத்துவமனை துப்புரவு பணியாளர்களிடம் நுரையீரல் பாதிப்பு உள்ளதா என்பதை ஸ்பைரோமெட்ரி மூலம் பரிசோதித்து மதிப்பீடு செய்தல்

ஆய்வு செய்யப்படும் இடம் :

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது : பங்கு பெறுபவரின் எண்.

இந்த ஆராய்ச்சிகளும் அதன் விவரங்களும் எனக்கு முழுமையாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட் விவரங்களை புரிந்து கொண்டு நான் என்னுடைய சம்மதத்தை தெரிவித்துக்கொள்கிறேன்.

இந்த ஆராய்ச்சியில் நான் என்னுடைய சொந்த விருப்பத்தின் பேரில் பங்கேற்கிறேன். இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின் வாங்கலாம் என்றும் அதனால் எவ்வித பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துக்கொண்டேன்.

நான் இந்த ஆராய்ச்சிக்காக ஸ்பைரோமெட்ரி மூலம் பரிசோதித்துக் கொள்ள சம்மதம் தெரிவிக்கிறேன்.

நான் இந்த ஆராய்ச்சியின் விவரங்களை கொண்ட தகவல்களை பெற்றுக் கொண்டேன். நான் என்னுடைய சுயநினைவுடனும் மற்றும் முழு சுதந்திரத்துடன் என்னை இந்த ஆராய்ச்சியில் ஈடுபடுத்திக் கொள்ள சம்மதிக்கிறேன்.

தேதி : கையொப்பம்.

MASTER CHART - SUBJECTS

SUBJECTS	Age	sex	Htincm	WtinKg	BMIKgm2	FVC	FEV1	FEV1FVC	FEF2575	PEF	Family size	Spirometric Pattern	Edu Sta (std)	Exp in Yrs
Vasantha	45	2	162	55	24.8	58	62	107	83	78	4	(Restrictive)2	5	>5
Alphonse	26	1	155	55	27.1	73	70	99	62	58	5	2	12	<3
Ambika	38	2	152	55	28.1	71	72	101	75	95	6	2	10	<3
Arutselvi	47	2	158	78	31.2	59	56	93	64	59	5	2	5	>5
Baby rani	38	2	155	40	16.6	41	36	89	60	51	6	2	10	>5
Baby	40	2	148	54	24.7	71	74	104	81	104	6	2	8	3-5Yrs
Chinnaponnu	42	2	150	62	27.6	59	61	103	68	59	5	2	5	>5
Deivarani	45	2	158	56	22.4	47	42	90	68	65	4	2	5	>5
Devi	43	2	148	50	22.8	76	76	101	75	96	5	2	5	<3
Dhanalaxmi	42	2	154	50	25.3	69	71	103	80	92	5	2	5	3-5Yrs
Kabil	45	2	148	50	22.8	75	73	98	67	54	6	2	5	<3
Kumari	45	2	149	45	20.3	54	49	90	58	65	5	2	5	>5
Deivanai	43	2	140	52	26.3	68	56	82	60	60	6	2	5	3-5Yrs
Lakshmi	46	2	144	45	21.7	57	57	99	55	48	5	2	3	3-5Yrs
Malliga	44	2	149	55	24.8	63	64	62	80	86	5	2	5	3-5Yrs
Manjula	39	1	160	56	21.9	46	58	121	150	82	5	2	10	>5
Mariammal	25	1	172	65	22.0	84	67	81	63	55	5	2	12	<3
Kannan	46	1	160	55	21.5	65	56	87	66	55	6	(Obstructive)3	UE	3-5Yrs
Pratap	32	1	180	63	19.4	52	43	81	67	58	5	3	10	>5
Rajam	38	2	142	42	20.8	70	73	106	90	117	6	2	8	<3
Ramesh	40	2	140	38	19.4	67	71	107	85	56	4	2	5	<3
Rita	42	2	155	58	20.4	64	65	101	70	99	6	2	5	3-5Yrs
Kumari	38	2	150	46	20.4	49	45	91	64	54	6	2	10	>5
Sagunthala	46	2	145	55	26.2	47	46	96	66	68	6	2	5	>5
Saraswati	44	2	150	53	23.6	72	72	101	78	111	6	2	8	<3
Shanthi	48	2	162	70	26.7	51	54	104	71	74	6	2	UE	>5
Sudha	40	2	152	60	26.0	58	60	103	74	67	6	2	8	>5
Sumithra	38	2	150	48	21.3	57	57	100	65	80	5	2	8	3-5Yrs
Thilagavathi	38	2	155	48	20.0	74	78	104	97	77	5	2	5	<3

Vasanthi	43	2	152	45	19.5	58	60	103	61	53	6	2	10	3-5Yrs
Vela	37	2	156	45	18.5	82	83	101	108	92	6	2	8	<3
Velankani	39	2	160	80	31.2	69	72	104	90	104	5	2	10	<3
Vinodh	36	2	153	60	25.6	64	59	93	56	85	5	2	12	3-5Yrs
Uma	32	2	161	64	24.7	71	68	97	68	75	5	2	12	<3
Dinesh	40	2	144	45	21.7	63	61	97	55	63	5	2	10	3-5Yrs
Mahalaxmi	45	2	142	40	21.7	65	63	98	67	84	6	2	5	<3
Savitiri	35	2	145	75	35.7	50	53	108	72	111	6	2	5	>5
Mariammaal	45	2	152	51	22.1	69	73	106	88	84	6	2	5	>5
Kanthimathi	42	2	154	51	25.7	68	69	102	74	97	6	2	5	>5
Sivagami	45	2	160	54	25.0	64	66	103	75	86	6	2	5	3-5Yrs
Sundari	41	2	158	58	27.2	65	67	102	77	81	6	2	5	>5
Gowri	41	2	152	56	24.2	65	69	106	79	88	6	2	5	<3
Paranjothi	39	2	160	58	26.6	61	63	103	75	83	5	2	10	>5
Gnanasoundari	40	2	148	52	23.7	72	75	104	80	94	5	2	10	<3
Selvi	32	1	150	35	15.6	76	71	93	65	78	6	2	12	<3
Sangeetha	25	1	163	70	26.3	69	72	106	93	60	5	2	12	<3
Savithiri	53	2	160	70	27.3	51	50	98	59	34	6	2	UE	>5
Neela	46	2	146	40	18.8	74	69	93	50	66	6	2	UE	<3
Rathi	42	2	150	72	32.0	59	56	94	52	58	5	2	8	3-5Yrs
Bagavathi	35	2	150	42	18.7	50	45	90	42	41	6	2	12	>5
Suguna	46	2	143	55	26.9	59	64	108	108	74	5	2	5	>5
Sridevi	38	2	150	46	20.4	49	45	91	44	42	5	2	12	<3
Sandya	41	2	150	52	23.1	72	73	101	73	93	6	2	8	<3
Latha	39	2	152	56	24.2	67	71	106	88	88	6	2	8	3-5Yrs
Geetha Rani	45	2	154	52	26.1	69	72	104	80	94	5	2	UE	3-5Yrs
Kayalvizhi	42	2	154	52	26.1	67	69	102	77	90	6	2	5	<3
Kala	29	2	175	52	20.2	68	59	88	47	80	6	2	12	>5
Srirangam	45	2	155	60	25.0	51	46	91	34	89	6	2	UE	>5
Udaya	40	2	158	45	18.0	41	33	80	35	29	7	2	10	>5

MASTER CHART - CONTROLS

CONTROLS	Age	sex	Htincm	WtinKg	BMIKgm2	FVC	FEV1	FEV1FVC	FEF2575	PEF	Family size	Spirometric Pattern	Edu Sta (std)	Exp in Yrs
Agala	43	2	150	55	24.4	63	64	102	74	91	7	2	10	NIL
Sasi	43	1	157	54	21.9	66	64	96	58	55	4	(Normal)1	12	NIL
Senthil	40	1	160	65	22.5	70	66	95	55	93	5	2	G	NIL
Shankar	44	1	152	50	22.2	119	115	100	80	84	5	1	G	NIL
Ganesh	45	1	155	54	18.7	100	95	96	75	79	6	1	G	NIL
Ramesh	43	1	158	67	21.2	76	80	105	85	88	5	1	G	NIL
Lakshmi	37	2	152	50	21.6	102	98	76	77	88	6	1	12	NIL
Rani	39	2	156	52	21.4	100	96	95	80	89	4	3	G	NIL
Charumathi	41	2	154	52	21.9	103	98	95	81	92	4	3	12	NIL
Agalya	43	2	158	52	20.3	97	94	99	85	79	3	1	12	NIL
Shruthi	37	2	155	76	24.8	86	87	100	88	93	3	1	G	NIL
Muniyammal	46	2	156	50	20.5	112	115	102	77	84	4	1	G	NIL
Nagammal	42	2	158	56	22.4	99	101	101	82	87	5	1	G	NIL
Alli	32	2	145	47	22.4	110	111	101	92	95	5	1	G	NIL
Nasreen	39	2	157	52	18.6	86	90	103	98	95	5	1	G	NIL
Nithya	36	2	155	56	20.6	57	58	100	65	79	5	2	G	NIL
Kalyani	48	2	156	55	22.6	67	71	106	85	82	5	1	G	NIL
Kamala	42	2	155	62	26.8	74	76	102	68	68	5	1	G	NIL
Sarasu	43	2	158	50	20.0	78	81	105	72	89	5	1	G	NIL
Malliga	39	2	152	48	20.8	98	98	100	80	79	5	1	G	NIL
Govindammal	43	2	156	56	23.0	98	100	101	84	80	6	1	G	NIL
Durga	44	2	150	58	22.7	94	98	103	79	76	4	1	G	NIL
Parameshwari	46	2	155	60	27.7	89	94	105	79	88	4	1	12	NIL
Raji	42	2	152	51	22.1	69	72	104	72	77	4	2	G	NIL
Revathi	42	2	156	50	20.5	108	114	105	79	78	3	1	10	NIL
Jeyanthi	44	2	156	57	23.4	108	114	105	75	80	4	1	12	NIL
Kalaiarasi	45	2	152	57	24.7	117	124	105	77	74	5	1	12	NIL
Selvi	46	2	155	60	27.7	78	83	107	85	82	6	1	G	NIL
Jeyalakshmi	43	2	155	50	21.9	105	110	104	76	83	6	1	G	NIL

Sameera	38	2	158	46	18.4	105	106	101	91	97	6	1	G	NIL
Lalitha	40	2	150	56	21.9	105	106	100	79	69	5	1	G	NIL
Shameemunisha	37	2	145	53	24.6	89	85	95	74	95	5	1	G	NIL
Sivesh	25	1	155	63	24.0	88	72	82	60	77	5	1	12	NIL
Gomathi	38	2	156	60	28.8	107	109	102	73	72	4	1	G	NIL
Renuga	40	2	152	60	26.7	99	103	103	94	99	3	1	12	NIL
Kokila	40	2	156	50	24.7	110	114	104	69	64	4	1	G	NIL
Kavitha	42	2	148	58	27.2	106	110	104	79	70	6	1	G	NIL
Malli	40	2	156	58	23.8	95	98	103	78	74	5	1	12	NIL
Komala	45	2	152	45	19.5	106	109	102	77	69	5	1	G	NIL
Mala	43	2	156	58	23.8	91	92	101	81	89	5	1	10	NIL
Sumathi	44	2	156	50	24.7	91	93	101	71	76	5	1	12	NIL
Fathima	30	2	152	70	30.3	102	98	97	85	79	4	1	G	NIL
Noorjahan	30	2	150	75	27.7	78	78	99	84	71	5	2	G	NIL
Jasmine	40	2	148	50	22.9	95	98	102	90	78	3	1	G	NIL
Jansi	44	2	152	50	26.0	111	111	100	97	74	4	1	G	NIL
Rosy	42	2	146	54	22.2	111	116	104	89	77	5	1	G	NIL
Veeramma	33	2	158	70	30.1	89	90	101	92	91	5	1	G	NIL
Pencillamma	34	2	160	75	27.7	86	88	101	82	74	5	1	G	NIL
Kaveri	27	2	155	59	22.5	84	84	99	88	97	6	1	G	NIL
Rangamma	45	2	162	58	25.9	101	107	104	85	88	6	1	G	NIL
Lakshmi 100	40	2	160	60	27.3	106	110	104	80	87	5	1	G	NIL
Ganga	40	2	155	55	24.8	103	108	105	86	88	5	1	10	NIL
Kamatchi	42	2	146	54	26.3	106	111	105	97	90	5	1	12	NIL
Dhanam	34	2	152	54	23.4	71	70	99	69	77	6	2	G	NIL
Paunu	43	2	154	58	24.5	67	69	103	74	80	5	2	G	NIL
Nithya	28	2	155	65	26.9	84	81	96	81	77	4	1	G	NIL
Vidya	33	2	152	58	25.9	92	91	98	80	73	5	1	G	NIL
Saroja	26	2	148	70	24.8	88	85	97	83	85	4	1	G	NIL
Uma	28	2	155	70	29.4	88	85	96	86	86	4	1	G	NIL
Maheswari	45	2	150	52	27.6	71	74	104	80	88	4	2	G	NIL
Visalatchi	45	2	157	58	27.6	66	68	102	72	88	3	2	G	NIL