Chapter 2

REVIEW OF LITERATURE
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The efficient talent identification procedures on athletes play a very important role in modern sports and has been a major factor in Olympic sports. The majority of talent identification is done at junior level in sports. Occasionally, individuals and small teams have to change their event or sports, after graduating to senior ranks because sports science tests have shown that these athletes have various physical and physiological capacities which may enable them to perform at an elite level in that particular sports as various sports have different energy demand. Thus athletes in younger ages are directed towards sports or particular events for which they are physically and physiologically best suited and obtain good results (Bloomfield, 1992).

The cardio-respiratory system is under severe stress during exercise. The ability to respond adequately to this stress is a measure of their physiological health. The increase in oxygen requirement during exercise can be assessed by exercise testing (Mrunal, Geetha, Shobha and Gouri, 1998).

Respiratory functions during exercise

An efficient pulmonary system is required by the athlete for the increased energy demands, imposed by the rhythmic muscular efforts during exercise (Dempsey, Gledhill, Reddan, Forster, Hamilton and Claremont, 1977). In many types of physical exercise, the respiratory frequency tends to become fixed to the exercise rhythm (Jasinskas, Wilson and Hoare, 1980). Bramble and Carrier (1983) pointed out that there is apparently a strict locomotor respiratory
coupling, especially in exercise where the stress of locomotion tends to deform the thoracic complex. Astrand and Rodahl (1986) later asserted that this strict—locomotor—respiratory coupling can be noticed in swimming, bicycle riding and running (Dejours, Mithoefer and Labrousse, 1957; Sipple and Gilbert, 1966; Astrand and Rodahl, 1986).

The water pressure on the thorax makes the respiration more difficult in swimmers. Astrand and Saltin (1961) and Holmer, Elliot, Saltin, Ekblom and Astrand (1974) observed that the respiration during swimming competition is well synchronised with the swimming strokes. During running, every footstep of athlete causes the diaphragm and abdominal contents to drop downwards which changes the synchronisation pattern of breathing in runners (Bursztyn, 1990).

Campbell (1964) pointed out that the rhythm, posture and movements during exercise affects the respiratory muscles. With increasing ventilation, the oxygen utilised for ventilation becomes progressively greater. So a certain percentage of O$_2$ uptake has to be met by the respiratory muscles to carry out respiratory functions during exercise (Otis, 1961 and Astrand and Rodahl, 1986). The respiratory muscles are activated through two types of motor neurons: (1) alpha ($\alpha$), which produces contraction of extrafusal muscle spindles of respiratory muscles and (2) gamma ($\gamma$) which activates the intrafusal muscle spindles. The $\alpha$- and $\gamma$-motor neuron system is linked with the afferent nerves of the muscle spindles which in turn elicits the reticular formation of brain during activation of muscles. Impulses from the reticular formation increase the $\alpha$- and $\gamma$-motor neuron activity which affects the respiratory muscles and results in increased thoracic volume.
The pulmonary ventilation increases during muscular exercise rectilinearly with the increase in O₂ uptake up to a certain level, after which the increase in ventilation becomes steeper. During exercise, pulmonary ventilation is more related to volume of CO₂ exhaled than to the O₂ uptake. During submaximal physical activity, the arterial Pco₂, Po₂ and H⁺ concentration are at the same level as at rest. During very heavy exercise, the anaerobic contribution to the energy yield is inevitably coupled with the production of H⁺ ions. Thus pH decreases and may become as low as 7.0 in the arterial blood. The relative hyperventilation that follows elevates the alveolar Po₂, but the arterial Po₂, drops towards lower value.
and $P_{\text{CO}_2}$ also drops to lower values. The lowering of arterial $P_{O_2}$ stimulates the breathing via peripheral chemoreceptors in the carotid and aortic bodies and sends impulses through sino-aortic mechanism to the brain stem. An increase in $P_{CO_2}$ and $H^+$ concentration also represents a stimulation leading to an increased ventilation but this effect is primarily elicited from medullary chemosensitive receptors, located on the ventral surface of medulla (Astrand and Rodahl, 1986). During exercise, the respiration increases by rate and depth proportionate to the concentration of $CO_2$ in blood. Depth of inspiration depends on actual number of motor units of inspiratory neurones that are firing along with their frequency of discharge whereas respiratory rate, depends on the length of time elapsing between firing (Nickol and Datta, 1994 and Mrunal et al., 1998).

The $\alpha-\gamma$ system has a possible tendency to produce hyperventilation, especially during the beginning of the exercise until the produced $CO_2$ has reached the lungs and $P_{\text{CO}_2}$ tends to drop. Through a negative feedback elicited from the respiratory centres, the $\alpha$ and $\gamma$ activity, driving the respiratory muscles may then be inhibited by the respiratory generators as $P_{\text{CO}_2}$ tends to drop. When the $CO_2$ reaches the lungs without being eliminated in sufficient quantity, the $P_{CO_2}$ of the arterial blood will rise and the inhibition of $\alpha$ and $\gamma$ system becomes diminished and results in hyperventilation (Astrand and Rodahl, 1986).

During exercise, the respiratory movements and respiratory frequency are adjusted according to the rhythm of exercise. The $\alpha-\gamma$ system activates a rhythmic co-ordinated switching between inspiration and expiration, partly determined by the rhythm of exercise. So $\alpha-\gamma$ system provides the synchronisation between the respiratory movements and rhythm of exercise for co-ordination of breathing (Von Euler, 1974). This combination of $\alpha-\gamma$ system makes it possible to
integrate respiratory and postural movements at the spinal level and to correct the “actual” length of respiratory muscles to the “wanted” length in accordance with the demands of breathing and also with change in posture during exercise (Astrand and Rodahl, 1986).

Energy demands of the body in athletes vary with different forms of athletic events. The O₂ requirement of the body to the various energy demands also differs in different disciplines of athletes. The locomotor-respiratory coupling with altered respiratory movements and respiratory frequency coincides with rhythm of exercise, results in alterations in respiratory functions during exercise. Pulmonary function test is one of the most important tools to measure the levels of physical capacity of a population (Singh and Sunderesh Peri, 1979). Physically fit athletes possess superior lung functions relative to less fit or sedentary subjects (Johnson, Reddan, Soar and Dempsey, 1981; Johnson, Reddan, Pegelow, Seow and Dempsey, 1991).

Increased VC and MVV of swimmers, skin divers and basket ball players have been reported following physical conditioning regimen. This shows that conditioning with different breathing patterns with more intensity for longer periods may elicit changes in lung volumes or functions (Bachman and Horvath, 1968; Lynch, Bove and Barrera, 1968).

Trained individuals show higher vital capacity than the untrained ones. It has been found that the level of physical activity can affect the ventilatory functions. Training of specific muscles for specific exercise may have gained better lung function (Kalyani Premkumar and Walter, 1994). Thus trained athlete can be distinguished from untrained counterpart with respect to the enlarged dimensions of respiratory functions which include VC, maximum flow rates and
pulmonary diffusing capacity (Astrand, Engstrom, Eriksson, Karlberg, Nylander, Saltin and Thoren, 1963; Holmgren and Astrand, 1966; Ekblom, Astrand, Saltin, Stenberg, Nallstorm, 1968, Hamilton and Andrew, 1976). Lung volumes and flow rates of young swimmers undergoing training were higher than their control subjects of similar age or height. Such changes are due to the breathholding exercise with increase in strength of respiratory muscles (Carey, Schaffer, Alvis, 1956; Astrand et al., 1963; Hamilton and Andrew, 1976; Ashaherwani, Desai and Solepure, 1989; Mohan Rao, Patel, Purohit, Kulkarni and Kashyap, 1993; Prateek Mehrotra, Narsingh Verma, Rajkumar Yadav, Sunita Tiwari and Neeraja Shukla, 1997). Continuous breathing pattern during swimming results in hypertrophy of the intercostals and diaphragmatic muscles. The external force exerted by the water pressure and repeated breathing during swimming alter the elasticity of lungs and chest wall in swimmers. The respiratory muscle hypertrophy and altered elastic properties together help the swimmers to improve their lung functions (Lakhera, Lazar Mathew, Rastogi and Sengupta, 1984).

The evaluation of the mechanical properties of lungs and chest wall can be made by determining maximum voluntary ventilation (MVV) (Astrand and Rodahl, 1986). MVV depends on the calibre of airways and efficiency of respiratory muscles and was found to be highest in runners and lowest in the basketball players. Boxing, basketball and gymnastics are comparatively less strenuous than swimming, football, running and wrestling and do not perhaps lead to much significant improvement of lung function of the individuals involved (Leith and Bradley, 1976; Lakhera et al., 1984).

Maximum voluntary ventilation in basketball, boxing, cricket, hockey and tennis players and FEV₁ values in football, hockey, swimming and volleyball
players are significantly higher than those of sedentary ones (Ghosh, Ahuja, Khanna, 1985). They were of the opinion that VC, MVV and FEV₁ in hockey players were found to be significantly higher than those of sedentary individuals. So it is clear from above mentioned experiments that athletic training has a significant effect on respiratory functions. The works of Newman, Smalley and Thomson (1961), Rash and Brant (1967) and Ness, Cunnigham, Eynon and Shah (1974) state that athletes have larger lung volumes and capacities than non-athletes of comparable age group. Superiority in pulmonary functions was observed in American athletes when compared with their Indian counterparts, due to the prolonged athletic training from early childhood to adolescence besides ethnic variations and overall dietary superiority (Lakhera et al., 1984).

James Kollias, Richard, Boileam, Larry Barlett and Elsworth (1972) were of the opinion that the athletic training does not result in higher lung functions and the respiratory functions are not affected during endurance exercise. Malhotra, Ramaswamy, Joseph and Sengupta (1972) showed that the measurement of FVC, FEV₁, MVV and PEF have not shown any difference between Indian athletes and non-athletes and hence the pulmonary function tests are not influenced by athletic status or physical training. Malhotra et al. (1972) observed that the resting respiratory volumes do not help to distinguish between athletes and non-athletes and the dynamic physiological functions during maximal physical effort are considered to be more important in this regard. A normal or decreased pulmonary functions were reported by Astrand and Rodahl (1986), Mc Ardle, Katch and Katch (1991) and Shephard and Astrand (1992) in athletes when compared with non-athletic counterparts.

Respiratory Muscle Fatigue During Exercise

The healthy human diaphragm can get fatigued, when one breathes at maximal pressure against an imposed external resistance (Roussos and Moxham, 1986). The inspiratory and expiratory muscles of the ribcage and abdominal muscles become highly active during mild exercise (Henke, Sharratt, Pegelow and Dempsey, 1988; Ainsworth, Smith, Eicker, Henderson and Dempsey, 1989; Dempsey, Johnson and Bayly, 1990a and Manohar, 1990). The study carried out by Johnson, Aaron, Babcock and Dempsey (1996) showed that although several other respiratory muscles are recruited with whole body exercise, the diaphragm is the primary inspiratory muscle and the most effective pressure generator for increasing alveolar ventilation and this provides the best index of respiratory muscle function. Mechanical consequences of these respiratory muscles during exercise include: (a) a dual action for both locomotion and respiration; (b) the reduction of end-expiratory lung volume by the activation of expiratory muscles, thereby placing the diaphragm at a long and more optimal length for tension.
generation; (c) the diaphragm operating more efficiently as a piston to increase
ribcage volume, because both the abdominal wall and ribcage are stiffened and
(d) the progressive increase of the expiratory muscle pressure development with
increasing exercise up to the point of the limitation of the expiratory flow, thus
maximum or near maximum expiratory flow-volume loop appears during maximal
exercise in the highly trained subject (Shephard and Astrand, 1992).

The volitional tests of peak inspiratory pressure, peak trans diaphragmatic
pressure or maximum sustainable ventilation have been used for the study of
whether prolonged strenuous exercise causes diaphragmatic fatigue (Loke et al.,
1982; Bye, Esan, Walley and Macklem Pardy, 1984; Hussain and Pardy, 1985;
Coast et al., 1990). But, such tests are not adequately controlled and are not
sufficiently objective of total body fatigue (Dempsey, Aaron and Martin, 1988).

A change in the frequency spectrum of the diaphragmatic electromyogram is
cited as evidence of diaphragmatic fatigue during short term exercise (Bye, Farkas
and Roussos, 1983; Sieck and Fournier, 1990). However, the validity of this index
remains speculative. The diaphragmatic pressure response to supramaximal
phrenic nerve stimulation provides objective evidence of diaphragmatic fatigue
(Bai, Rabinovitch and Pardy, 1984; Bellemare and Bigland-Ritchie, 1987;

During exercise, the increased respiratory muscle demand,
associated with increase in intensity and frequency of contraction and the decreased
respiratory muscle capacity, results in ‘stress’ to inspiratory muscle (Leblanc,
Summers, Inman, Jones, Campbell and Killian, 1988). Mador and Acevedo
(1991b) observed that fatigue of respiratory muscles and increase in minute
ventilation are due to increased breathing frequency during exercise. Fatigue of
inspiratory muscles limits the ability to sustain a high minute ventilation and this may be an important factor limiting exercise for the decreased lung function (Mittman, Edelmon, Norris and Shock (1965); Zoeche, Fritts and Courmand, 1960; Jones, Jones and Edwards, 1971; Devenne, Macklem and Roussos, 1978; Grassino, Gross, Macklein, Roussos and Zagelbaum, 1979; Robertson, 1982; Belman, Michael and Glenn, 1988).

The works of Leblanc et al. (1988) clearly show that after an endurance race, athlete showed a reduction in PEFR and FEV₁ than before the race which was due to muscle fatigue. They further reiterate that there was no evidence that fatigue limits track performance. Martin, Bruce and Stager (1981) suggested that respiratory muscle fatigue does not ‘impair’ the performance of athlete. Loke et al. (1982), Martin, Heetzelman and Hsiung-ing Chen (1982) and Bye et al. (1984) were also of the same opinion that respiratory muscle fatigue is evidently a consequence of marathon running in which both the strength of respiratory muscles and MVV declined following the race.

The increased energy demands during exercise could limit respiratory functions in athletic performance. The energy demand of the respiratory muscles to increase the pulmonary ventilation, necessitates a marked increase in the oxygen consumption. An increase in pulmonary ventilation beyond a certain point would not be physiologically useful, since all the additional O₂ gained would be required for breathing (Otis, 1964; Bye et al., 1984). So the fact is that the O₂ uptake reaches a distinct plateau during extensively heavy exercise even if the rate of exercise and pulmonary ventilation is further increased. Therefore, the net effect of an extra respiratory effort may be questioned (Astrand and Rodahl, 1986; Bursztyn, 1990).