DO CARDIORESPIRATORY FREQUENCIES SHOW ENTRAINMENT WITH HOPPING IN THE TAMMAR WALLABY?

BY R. V. BAUDINETTE, B. J. GANNON, W. B. RUNCIMAN, S. WELLS

Schools of Biological Science and Medicine, The Flinders University of South Australia, Bedford Park, South Australia 5042, Australia

AND J. B. LOVE

Department of Anaesthesia, Repatriation General Hospital, Heidelberg, Victoria, Australia

SUMMARY

Breathing, heart and gait frequencies, tidal volume, cardiac output, and rates of oxygen consumption were measured in tammar wallabies (*Macropus eugenii* Desmarest) hopping on a treadmill. At speeds greater than $1.6 \, \mathrm{m \, s^{-1}}$ the rate of metabolic power consumption was independent of hopping speeds. Blood lactate levels within the speed range where $\dot{V}_{O_2}$ was independent of speed showed a mean increase of $4.8 \, \mathrm{mmol \, l^{-1}}$. During bipedal hopping, the frequencies of breathing and limb movement are phase-locked in the ratio of $1:1$. Inspiration begins as the animal leaves the ground and may be a passive process driven by a visceral piston. A relatively large central tendon in the diaphragm may correlate this function. Unlike breathing frequencies, cardiac frequencies show no entrainment with hopping. The site of dissipation of the presumed large arterial pressure excursion is unknown.

INTRODUCTION

The rate of metabolic energy consumption during hopping in a kangaroo is independent of speed, a phenomenon which has been explained by the storage of elastic strain energy in muscles and tendons, and its conversion to gravitational potential energy and kinetic energy during phases of the step cycle (Dawson & Taylor, 1973; Alexander & Vernon, 1975; Morgan, Proske & Warren, 1978; Alexander, 1984). There are other small mammals that move bipedally (see Baudinette, Nagle & Scott, 1976; Thompson, MacMillen, Burke & Taylor, 1980; Biewener, Alexander & Heglund, 1981), but treadmill studies have shown that the costs of locomotion for these species are equivalent to those of similarly sized quadrupeds (Thompson *et al.* 1980). Marsupials of the family Macropodidae are the only large hopping animals and, among mammals which move in this way, the only ones which show an uncoupling of speed and metabolic rate.

The present study investigates cardiorespiratory correlates of hopping and examines the hypothesis that the simple in-phase movement of the hind limbs of macropods shows entrainment with breathing and heart frequencies. The animal used was the tammar wallaby, *Macropus eugenii*, a small (5 kg) bipedal marsupial which now occurs mainly on islands off the southern Australian coast.

MATERIALS AND METHODS

Twelve adult tammar wallabies were collected under permit from Kangaroo Island, South Australia. They were housed in large outdoor enclosures, where their grazing was supplemented with commercial food pellets and lucerne. The body masses of the animals were stable during the experiment and ranged from 4·25 to 5·10 kg. Over a period of 4–5 weeks the animals were trained to hop on a motor-driven treadmill 1·4 m long and 0·6 m wide, in which the speed could be continuously varied with a hydraulically driven assembly. The belt speed indicator of the apparatus was calibrated from the length of the belt and the time for a sample of revolutions.

Rates of oxygen consumption ($\dot{V}_{O_2}$) were measured using an open-flow system. The animals wore lightweight masks through which air was metered at rates around $0·3 \, \mathrm{l\,s^{-1}}$ (STPD). The difference in fractional oxygen content between room air and the air leaving the mask was measured with a Servomex (model OA 184) para-magnetic oxygen analyser. Steady-state conditions of oxygen consumption were considered to be those showing less then 3 % variation over a period of 3 min. The entire system was calibrated using a metered flow of nitrogen in a manner similar to that described by Fedak, Rome & Seeherman (1981). No reduction in measured oxygen consumption or simulated oxygen consumption using metered nitrogen was detected when the flow rate was reduced by 30 % and we assumed that no spillage of respiratory gases occurred from the mask. In calculations of power input, we have assumed a respiratory quotient of 1·0; errors in calculated $\dot{V}_{O_2}$ derived from variation in this factor will be less than 1 %. In three individuals, blood samples were taken from a lateral tail vein while the animals were at rest and after hopping at $4 \, \mathrm{m\,s^{-1}}$ for 5 min. The lactate concentrations in $50 \, \mu l$ subsamples were determined using a commercial kit (Boehringer Mannheim).

Measurement of hopping and breathing frequencies

A capacitive transducer glued to shaved skin above the sacrum was used to measure hopping frequencies. A thin wafer of piezoelectric material (Brush multi-morph) was mounted in a cantilever beam 1 cm in length and heavily damped with a small quantity of silicon grease. The assembly was sealed in a Perspex capsule and connected *via* a light coaxial cable to a charge amplifier.

To determine the phase lag of the accelerometer it was mounted, together with a microswitch, above a rotating cam which could be driven at frequencies up to 6 Hz. The output from both was fed to a dual-beam oscilloscope. At strike frequencies up to 4 Hz, the response of the accelerometer was within 5 ms of that of the microswitch,

showing that the former could accommodate the maximum hopping frequency with negligible phase lag. The amplifier was virtually independent of the shunt capacitance across the input and the long (2 m) shielded cable between the transducer and the preamplifier did not affect the response. The amplified signal was fed to a Grass 7P511 amplifier and then to one channel of a Grass polygraph.

An impedance pneumograph was used to monitor the rate of respiration (f_B) and provide a measure of tidal volume (V_T). The instrument consisted of a 30-kHz oscillator which provided a small alternating current through two platinum electrodes glued to each side of the animal's thorax. A reference electrode was attached to the back of the animal. The output from the instrument was amplified (Grass 7P511) and fed to a second channel of the polygraph. To establish that the measured impedance changes were due to respiration, a fine thermocouple was fixed near the animal's nostril and temperature changes were correlated with impedance.

The pneumograph was calibrated in two anaesthetized wallabies. A cuffed tracheotomy tube was inserted *via* a midline neck incision under anaesthesia (2 % halothane in air) and the cuff inflated so that no gas leaked from the breathing circuit. A 250 ml calibrated Hamilton syringe was used to inflate and deflate alternately the lungs with a series of different gas volumes and the resulting changes in impedance between reference points on the chest wall were recorded on a storage oscilloscope. The maximum values of impedance during inspiration showed a reproducible curvilinear relationship with the volume of gas injected.

Measurement of heart rate (f_H)

Intracardiac electrocardiographic recordings were used to monitor heart rates, since surface recordings were obscured by pectoral and intercostal muscle activity. Under halothane anaesthesia and fluoroscopy, a sterile probe [quadrupolar transvenous electrocardiograph probe (USCI, Billerica, MA, USA)] was introduced *via* the left jugular vein and left anterior vena cava into the right atrium. Following surgery, the external portion of the lead was fastened to a collar around the neck and the animal was given 4 days for recovery before any experiments were begun. Intracardiac electrocardiogram (ECG) recordings were amplified (Grass 7P511) and displayed on a physiograph recorder (Grass Instruments, Quincy, MA, USA).

Measurement of cardiac output

Cardiac output ($\dot{V}_B$) was measured in resting and moving animals by a thermal dilution technique. The method was first tested on anaesthetized animals against a standard dye dilution method (see below). A Swan–Ganz catheter (5F, Edwards Laboratories, Santa Ana, CA, USA) was introduced into the left jugular vein in four anaesthetized animals. Using an X-ray apparatus equipped with an image intensifier (Toshiba model SXT-6-6), the catheter was positioned through the left anterior vena cava, coronary sinus and right ventricle into the pulmonary artery (Love *et al.* 1985). The catheter was secured in a small pack attached to the animal's back and it was left to recover for at least 4 days.

For animals at rest in a covered cage or while hopping on the treadmill, volumes of 5 ml of ice-cold isotonic saline were injected *via* the proximal port of the catheter and the resultant thermal signal was sensed by the more distal thermistor. Using the image intensifier, we had previously demonstrated that the injected saline left the catheter from a port in the right atrium or in the left anterior vena cava close to the right atrium. Integration of the area under the thermal dilution curve and computation of the cardiac output was carried out by a cardiac output computer (model 9550, Edwards Laboratories); a form of the Stewart–Hamilton equation, involving the volume of injected fluid, the initial blood temperature and the specific heats of saline and blood, was used to integrate the resultant time–temperature thermodilution curve (Ganz & Swan, 1972).

The validation of this technique using the dilution of a dye was carried out in three wallabies; this validation procedure has been described previously in detail (Runciman, Illsley & Roberts, 1981a). The animals were anaesthetized with halothane; initially *via* a hand-held mask, and then *via* a cuffed endotracheal tube. Indocyanine green (Hynson, Westcott & Dunning, Baltimore) was made up to a final concentration of $2 \cdot 5$ mg ml^{-1} in isotonic saline and volumes of $0 \cdot 5$ ml were injected *via* the proximal port of the Swan–Ganz catheter. Arterial blood was sampled *via* a catheter in the descending aorta by a syringe pump (Sage model 367; Waters, Minnesota) at a flow rate of 20 ml min^{-1}. A densitometer cuvette (Model dc-410, Waters), placed between the catheter and the syringe, was used to measure the concentration of the dye, and the resultant signal was amplified on a cardiac output analyser (Model Td-1A, Waters) and fed to a potentiometric recorder. The system was calibrated using samples of wallaby blood to which known quantities of the dye had been added. Using the resultant calibration factor, cardiac output was calculated from the area under the dye-dilution curve. Forty-three paired thermal dilution and dye-dilution measurements of cardiac output were made in the three animals. By altering the level of anaesthesia, cardiac outputs were varied over a four-fold range.

The relationship between determinations using dye dilution and thermal dilution was analysed by a least-squares analysis, resulting in the equation:

$$y = 0 \cdot 96x + 0 \cdot 44,$$

where y is the cardiac output determined by dye (in l min^{-1}) and x is the corresponding value from thermal dilution. The correlation coefficient (r) was $0 \cdot 85$, the standard errors of the intercept and the slope were $0 \cdot 05$ and $0 \cdot 96$, respectively. Reasons for the positive intercept on the y axis (loss of thermal indicator) have been discussed in detail elsewhere (Runciman, Illsley & Roberts, 1981b).

The morphometry of the diaphragm

The diaphragms were excised from four tammar wallabies and the wet masses determined. The costal and crural portions of the musculature and the central tendon were outlined by tracing their boundaries on drawing paper with a sharp point and the relative areas were measured using a digitizing pad, Apple II+

computer and associated software (Stereometric Measurement and Analysis; Scientific Microprograms, Raleigh, NC).

Experimental protocol

In animals of this size it was not possible to obtain from each animal simultaneous measurements of oxygen consumption, cardiac output, tidal volume, heart and breathing rates, and gait patterns. The experiments were conducted in the following sequence. (i) Measurement of $\dot{V}_{O_2}$ in five animals trained to wear a mask. (ii) Simultaneous measurement of breathing, heart and gait frequencies in the same group of animals. During these experiments, the animals would move at higher speeds than when wearing a respiratory mask, but at low speeds gait and respiratory frequencies were irregular since the animals moved relative to a fixed position on the treadmill. (iii) A second group of four animals was used for the determination of cardiac output at rest and at two speeds at either end of the range over which we could measure $\dot{V}_{O_2}$. Levels of $\dot{V}_{O_2}$ were recorded from the same animals at equivalent speeds but on the following day. Members of this group were later killed to verify the absence of damage due to catheter placement and the diaphragms were removed for measurement.

RESULTS

Power input and gait patterns during hopping

At speeds between 1·25 and 1·6 m s^{-1} the wallabies changed gait from a 'pentapedal' form, using all four limbs and the tail, to bipedal hopping (Fig. 1). Up to 1·6 m s^{-1}, $\dot{V}_{O_2}$ increased linearly with speed, the relationship being described by the equation:

$$y = 0\cdot32x + 0\cdot38 \ (r = 0\cdot71),$$

where y is $\dot{V}_{O_2}$ (in ml s^{-1} kg^{-1}) and x is speed (in m s^{-1}). The regression-derived line relating these variables at speeds between 1·6 and 4·8 m s^{-1} (y = 0·025x + 0·89) has a slope which is not significantly different from zero ($P = 0\cdot08$). The mean plateau level of $\dot{V}_{O_2}$, 0·89 ml s^{-1} kg^{-1}, is 12·5 times the expected basal or standard level (Dawson & Hulbert, 1970) and about eight times the level measured from animals resting on the treadmill. In animals previously at rest, blood lactate levels showed a mean value of $3\cdot4 \pm 1\cdot7$ mmol l^{-1} ($\pm$s.e., $N = 3$). In the three individuals after 5 min exercise at 4 m s^{-1}, the mean level had risen to $8\cdot2 \pm 1\cdot4$ mmol l^{-1}.

Hopping frequency was independent of speed (Fig. 1); the line relating these variables (y = 0·09x + 2·84, $r = 0\cdot71$) has a gradient not significantly greater than zero. Increases in speed were dependent on changes in stride length; regression analysis of these variables results in the line:

$$y = 0\cdot26x + 0\cdot204, \ r = 0\cdot98.$$

In both equations, x represents the speed (in m s^{-1}) and y is hopping frequency and stride length, respectively.

Respiratory frequency and tidal volume

The frequency of breathing and the frequency of limb movement showed a 1:1 coupling at all treadmill speeds (Fig. 2). The relationship between these variables is described by the equation:

$$y = 0 \cdot 98x + 0 \cdot 04 \ (N = 47, \ r = 0 \cdot 99).$$

Moreover, the relationship between different phases of the locomotory and respiratory cycles is fixed. Inspiration begins as the animal leaves the ground and continues through about 26 % of the hopping cycle (Fig. 3).

The values for tidal volume, breathing rate and the calculated minute volumes are given in Table 1. Tidal volumes at hopping speeds of 2·5 and 4·5 m s^{-1} were statistically similar and represent a 50 % increase over levels recorded from animals
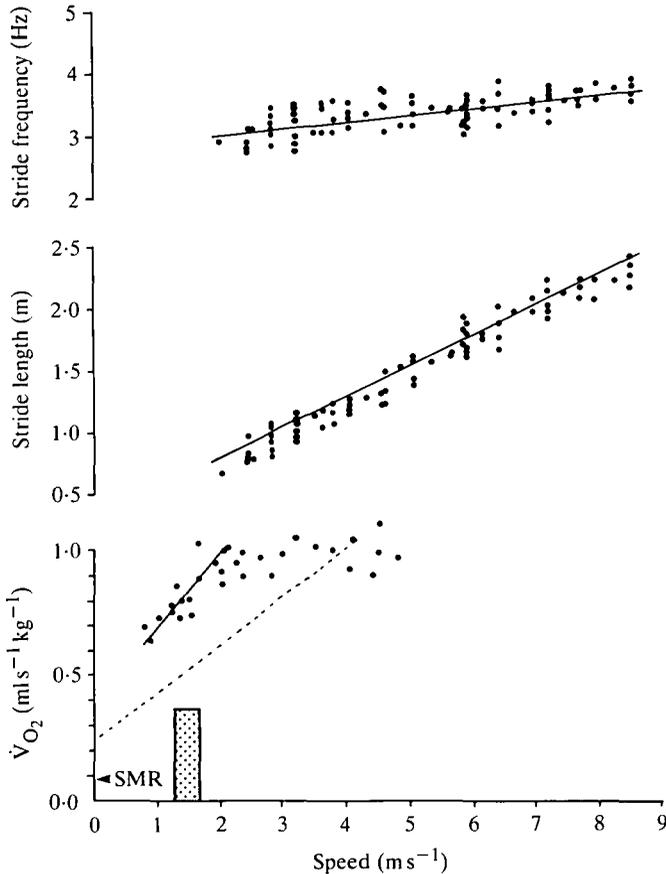

Fig. 1. Steady-state oxygen consumption, stride length and stride frequency plotted as a function of speed in five tammar wallabies. The shaded speed range indicates the transition between quadrupedal and bipedal movement. The equations describing the least-squares regression lines are included in the text. The dotted line indicates the expected relationship between $\dot{V}_{O_2}$ and speed for a quadruped of equivalent body size (from Taylor, Heglund & Maloiy, 1982). SMR, standard metabolic rate for the tammar wallaby (Dawson & Hulbert, 1970).
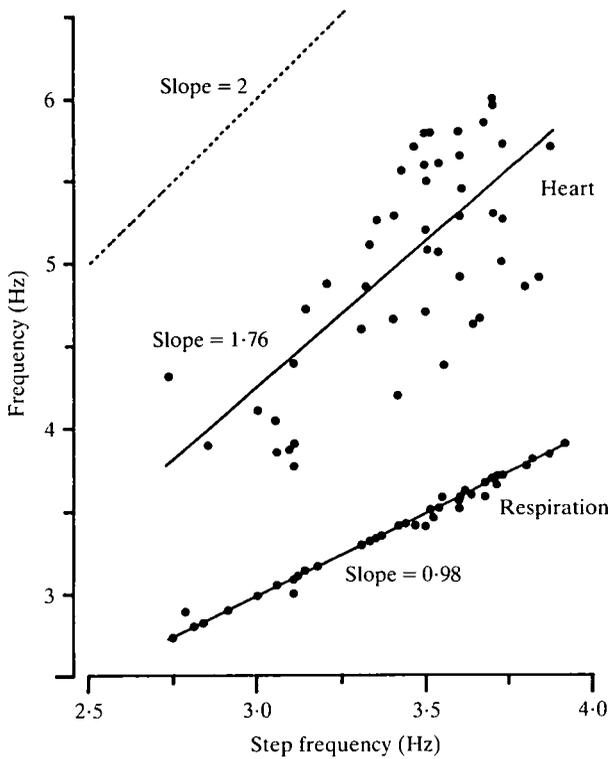
Fig. 2. Respiratory and heart frequencies plotted as a function of hopping frequencies in tammar wallabies. The equations describing the lines of best fit are given in the text. The dotted line indicates the theoretical coupling ratio of 1:2.
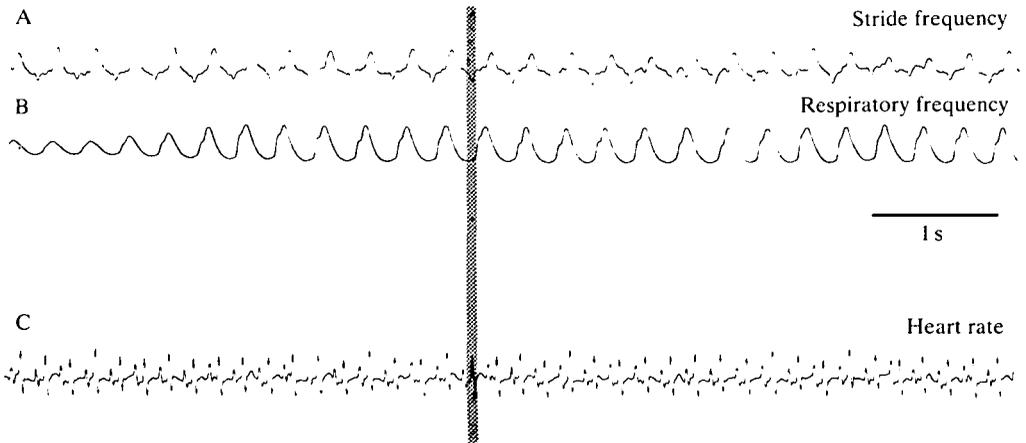

Fig. 3. Portion of a sequence of a tammar wallaby hopping at $4.0\,\mathrm{m\,s^{-1}}$ showing stride frequency (A), respiratory frequency (B) and heart rate (C). In B a positive upward gradient indicates inspiration, a negative gradient indicates expiration. The shaded portion in A indicates, for one step cycle, the time for which the animal is in contact with the ground.

Table 1. *Rates of oxygen consumption and cardiorespiratory parameters in tammar wallabies at rest and at two hopping speeds*

Speed (m s^{-1})	$\dot{V}_{O_2}$ (l kg^{-1} min^{-1})	Heart rate (min^{-1})	Stroke volume (l)	Cardiac output (l min^{-1})	Breathing rate (min^{-1})	Tidal volume (l)	Minute volume (l min^{-1})
Rest	0·0058 ± 0·0001	195 ± 15	0·0064 ± 0·05	0·61 ± 9·0	97·2 ± 0·01	0·050 ± 1·05	4·76
2·5	0·0626 ± 0·003	240 ± 21	0·0062	1·49 ± 0·10	210·0 ± 16·2	0·074 ± 0·02	15·54 ± 4·28
4·5	0·0634 ± 0·004	289 ± 27	0·0063	1·83 ± 0·10	196·5 ± 12·4	0·078 ± 0·02	15·43 ± 3·01

Values are means ± S.E., $N = 5$.

resting on the treadmill under similar conditions. The three-fold increase in minute volume from the resting condition compares with the approximately ten-fold increase in oxygen consumption.

Heart rate

Heart rates (y) increase significantly with hopping speed (x) ($y = 0 \cdot 27x + 3 \cdot 48$, $N = 46$, $r = 0 \cdot 67$) (Fig. 4). There was no evidence of entrainment between cardiac frequency and either hopping or breathing frequencies (Figs 2, 4). The relationship between these variables (x) and heart rate (y) is linear:

$$y = 1 \cdot 76x - 1 \cdot 02 \ (N = 46, \ r = 0 \cdot 67).$$

Cardiac output

Measurements of $\dot{V}_{O_2}$ and cardiac output, together with the derived values of stroke volume, are shown in Table 1. Cardiac output showed a significant ($P < 0 \cdot 05$) increase when speed increased from 2·5 to $4 \cdot 0 \, m \, s^{-1}$; this was a consequence of a significant increase in heart rate rather than stroke volume. Comparing the mean values of $\dot{V}_{O_2}$ and cardiac output from resting to exercise values shows that arteriovenous oxygen content differences must increase at least three-fold during hopping.

Diaphragm morphometry

Areas and mass of the whole diaphragms and their muscle components are shown in Table 2. The expected values are from a recent allometric analysis by Leiter,
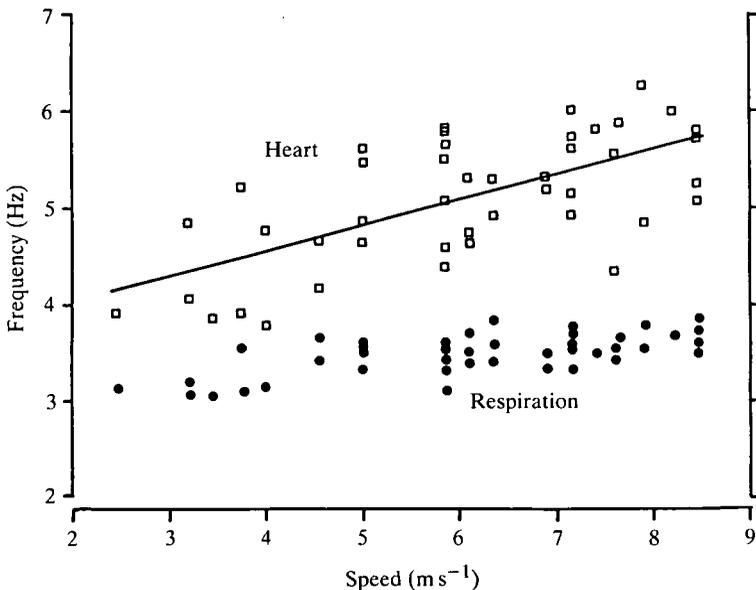

Fig. 4. Heart rate (open squares) and respiratory rate (closed circles) plotted as a function of speed in five tammar wallabies. The equations relating the measured parameters to speed are given in the text.

Table 2. *The mass and the areas of the diaphragms from four tammar wallabies of mean body mass 4·7 kg*

	Tammar wallaby	Expected value
Mass of whole diaphragm (g)	$12·79 \pm 0·96$	$21·8$
Mass of costal and crural muscle (g)	$11·56 \pm 0·47$	$19·4$
Area of diaphragm (cm^2)	$109·8 \pm 7·4$	$117·8$
Area of costal muscle (cm^2)	$47·9 \pm 5·1$	—
Area of crural muscle (cm^2)	$14·0 \pm 3·0$	—
Area of central tendon (cm^2)	$48·2 \pm 1·6$	—

The expected values are from the allometric equations of Leiter, Mortola & Tenney (1986). Values are means ± S.E.

Mortola & Tenney (1986). The wallaby diaphragm is of similar area to that seen in mammals of equivalent body mass, but is lighter, presumably due to its larger area of tendon relative to muscle.

DISCUSSION

The relationship between $\dot{V}_{O_2}$ and speed in the tammar wallaby shows the same phenomenon of uncoupling of oxygen demand and speed as previously demonstrated in the red kangaroo by Dawson & Taylor (1973). Blood lactate levels within the plateau region of $\dot{V}_{O_2}$ are well below those seen in mammals at maximum levels of oxygen consumption (see Seeherman, Taylor, Maloiy & Armstrong, 1981). At lower speeds, $\dot{V}_{O_2}$ increases with speed are more marked than in quadrupeds of an equivalent size, the line relating $\dot{V}_{O_2}$ (y) with speed (x) ($y = 0·32x + 0·38$, $r = 0·71$) having a slope significantly greater than that of $0·24$ seen in a dog of equivalent body mass (Taylor, Heglund & Maloiy, 1982).

Gait and respiratory frequencies are phase-locked in the simple ratio of 1:1 in the tammar wallaby and there appears to be no other coupling ratio available to the animal. In man, the other bipedal animal where equivalent data are available, switches of ratio are observed, with 2:1 being dominant, but at least five others may be observed (Bramble, 1983; Bramble & Carrier, 1983). In the few quadrupedal mammals examined, locomotory–respiratory ratios of 1:1 are widespread, although horses and jackrabbits have been observed at different ratios (Bramble & Carrier, 1983).

The coordination of lung ventilation with the movement of limbs also occurs in birds. A tight phase-locking between wing beat and respiratory frequencies has been demonstrated, with ratios of 1:1 in pigeons (Hart & Roy, 1966), 3:1 in barnacle geese (Butler & Woakes, 1980) and 5:1 in ducks and pheasants (Berger, Roy & Hart, 1970).

The situation in the tammar appears to be simple. When the rear limbs leave the substrate the rib cage begins to accelerate with respect to the abdominal organs. The phase difference of these two cyclic events may be increased by the compliant nature of the diaphragm and the abdominal wall. The resultant change in thoracic volume

initiates inspiration. Anatomical evidence to support such a hypothesis is the relatively large and thin central tendon of the diaphragm. At a gait frequency of 2·8 Hz, peak inspiration occurs 80 ms after the animal leaves the ground and an average inspiration is completed at about 26 % of the gait cycle. When the animal reaches the highest point of the hop, the expiratory phase is about 30 % complete. The concept that the visceral mass, slightly out of phase with the musculoskeletal framework but coupled *via* the diaphragm, can act as a 'visceral piston' was first proposed by Bramble & Carrier (1983). Their idea is most simply illustrated by the hopping wallaby. Steady electrical activity in intercostal muscles indicating a rigid thorax and a diaphragm comprising an unusually large proportion of central tendon correlate with such a simple piston arrangement.

There is an interesting difference in the relationship between oxygen consumption and lung ventilation in man and kangaroos which may relate to the ability of man to vary locomotory–respiratory ratios. Lung ventilation and oxygen demand increase linearly with speed in man (see e.g. Margaria, 1976), but both increase at a greater rate than stride frequency. Bramble (1983) has suggested that if a single coupling ratio only were available, large increases in tidal volume would be necessary to meet oxygen demand. A change in coupling ratio may be initiated by an approaching limit to tidal volume. In wallabies no such problem exists. Due to storage of energy in muscle and tendon and the uncoupling of metabolic demands and speed, ventilatory demands can always be met by a relatively constant tidal volume at a breathing frequency which is phase-locked to locomotory frequencies.

The simplest model to explain the coupling ratios seen in wallabies is that the passive increase in intrathoracic volume is the direct result of a visceral piston acting on the diaphragm. What is the relationship between this locomotory oscillator and the pontomedullary respiratory centre? This question has been recently addressed by several studies which are reviewed by Eldridge, Millhorn, Kiley & Waldrop (1985). While a feed-forward or 'central command' hypothesis enjoys good experimental evidence as the primary drive of exercise hyperpnoea, reflexes arising from lung inflation or input from the diaphragm may underlie the marked entrainment such as seen in tammar wallabies. The recent demonstration of respiratory phase-locking during mechanical ventilation in humans provides an analogy (Graves *et al.* 1986).

Most of the muscle mass of macropods moves the hind limbs, and it seems likely that most of these muscles in kangaroos and wallabies will be in an active state when the animal lands. This situation contrasts with that in quadrupeds, in which some muscle systems would be relaxed while the contralateral ones were contracted. During the time of maximal muscle contraction in macropods, the vascular impedance would be expected to be extremely high and it seems a reasonable hypothesis that locomotory and heart frequencies would be phase-locked to reduce the resultant excursions in arterial pressure. Potentially this would appear to be of particular importance in macropodid marsupials in which the shape of the animals and their distribution of muscle mass results in wave reflections which have a greater effect on aortic pressure pulse than that observed in any other animal (Avolio, Nichols & O'Rourke 1984, 1985). Our results clearly show that there is no evidence of

entrainment of cardiac and gait frequencies; the former increases linearly with hopping speed, while gait remains at a constant frequency. The site of resolution of large pressure excursions, resulting from shape-dictated discrete reflecting sites and a presumed marked increase in vascular impedance as the animals land, is unresolved. The compliance properties of arteries from kangaroos are unknown and there is no evidence of expanded aortic bulbs as seen in some diving mammals.

Clearly there are several correlates of bipedalism in cardiorespiratory function. The storage of energy in elastic structures and the resulting uncoupling of power demands and speed has produced a respiratory system in which lung ventilation may be a largely passive process driven by a visceral piston. A reduced energy cost for locomotion has also removed the necessity for variable locomotory–respiratory gearing. Bipedalism may also result in novel adjustments of the cardiovascular system and studies are currently under way to resolve this question.

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