Jevning, R., A. F. Wilson, J.P. O'Halloran, and R.N. Walsh. Forearm blood flow and metabolism during stylized and unstylized states of decreased activation. American Journal of Physiology 245:R110-R116, 1983.

Forearm blood flow and metabolism during stylized and unstylized states of decreased activation

R. JEVNING, A. F. WILSON, J. P. O'HALLORAN, AND R. N. WALSH
Departments of Medicine, Physiology, and Psychiatry, University of California, Irvine, California 92717

JEVNING, R., A. F. WILSON, J. P. O'HALLORAN, AND R. N. WALSH. *Forearm blood flow and metabolism during stylized and unstylized states of decreased activation.* Am. J. Physiol. 245 (Regulatory Integrative Comp. Physiol. 14): R110–R116, 1983.—We have measured forearm oxygen consumption and blood flow changes during two wakeful rest behaviors. We have observed acute reduction of forearm respiration (28%) during an acute stylized rest state (TM) and a nonsignificant small decline (11%) during unstylized ordinary eyes-closed rest. These changes were not associated with significant change of forearm blood flow or glycolytic metabolism. Hence, forearm oxygen consumption decline was due almost solely to decreased rate of oxygen extraction. Small variation of forearm blood flow implies that little of the previous findings of increased nonrenal, nonhepatic circulation during TM or increased nonrenal circulation during ordinary rest can be accounted for by altered muscle blood flow, which therefore is consistent with possible increased cerebral blood flow. However, reduced muscle metabolism was a likely contributor to the forearm metabolic decline. The lack of coupling between metabolic and blood flow changes during TM indicates limitation of obligatory coupling between cardiovascular and metabolic function in the rest state of TM.

behavior; relaxation; transcendental meditation technique; muscle and skin blood flow; lactate generation; oxygen consumption

CURRENT UNDERSTANDING of metabolic and cardiovascular changes associated with behavioral states is based primarily on study of states of increased activation such as defense or stress (5, 7, 11, 13), exercise (13, 24, 32), or differing attentional demands (33). In particular, several investigations have focused on limb blood flow (1, 5, 7, 33), metabolism (5), heart rate, and blood pressure (32, 33). Models have been proposed for interrelationship of cardiac and metabolic changes; e.g., Obrist and colleagues (24) have hypothesized that cardiovascular changes represent adjustments of cardiac output and its distribution to meet metabolic demands in most, if not all, behavioral states.

Few cardiovascular and attendant metabolic data exist, however, on states of acutely decreased activation, although regular elicitation of rest-relaxed states is now common (15, 20, 31). For study of cardiovascular and metabolic changes and their interrelationship at this end of the activation spectrum, the mental technique known as "transcendental meditation" (TM) is convenient, since rapid metabolic and cardiovascular changes consistent with decreased activation have been extensively described during this behavior (4, 12, 19, 20, 31), and there exists a relatively large and homogenous body of individuals who have been regularly eliciting this state twice daily for periods of 30–40 min over the course of several years (31). Practiced while seated comfortably, the technique allegedly requires no physical or mental control and is enjoyable and easily learned (31). Some of the physiological changes previously described include (31), acute decrease of red cell metabolism (17), increased frontal and central alpha activity in the electroencephalograph (4), and acute decline of adrenocortical activity (16); blood pressure and body temperature do not change acutely (31).

A recent study in this laboratory (19) also indicated significant increase (44%) of that fraction of the circulation that is nonrenal and nonhepatic. Hence, although it has been hypothesized that there is a contribution of muscle relaxation and concomitant decline of metabolic rate (12) to the decline of total metabolic rate during rest-relaxation states, the large increase of nonrenal nonhepatic flow (19) suggests the possibility of substantially increased muscle blood flow (19). Because of these conflicting ideas and because direct measurement of metabolic and circulatory activity of muscle during relaxed behavioral states has not been reported, we have studied relative forearm blood flow and oxygen consumption changes during TM. Also, because a comparable decline of total metabolic rate (12) and a small increase (12%) of nonrenal nonhepatic blood flow have been reported during ordinary unstylized eyes-closed rest-relaxation (19), subsequently denoted as "R," relative forearm blood flow and oxygen consumption were also measured during this behavior.

Inasmuch as muscle and skin blood flow comprise approximately 25% of nonrenal nonhepatic blood flow at rest, and cerebral blood flow the bulk of the remainder (13), the previously observed increments of nonrenal nonhepatic blood flow were due to significant increases of skin and muscle blood flow (and, possibly, metabolism) and/or increased brain blood flow. The goal of the present research was to determine more precisely the possible specific tissue contributions to the blood flow changes and to the overall decline of metabolic rate noted during these rest states and the relationship between the metabolic and blood flow alterations. The data may also help

elucidate more precisely the relationship between, and significance of, different means of rest state induction.

METHODS

Arteriovenous difference of oxygen and lactate content and relative change of pulsatile blood flow were monitored in two separate groups of subjects: 32 normal, lean, young adults (10 women, 22 men, ages 25–35) who were long-term practitioners of TM (that is 4–5 yr of regular elicitation for 30- to 40-min periods twice daily) and 25 individuals of similar background (7 women, 18 men, ages 21–32) who had no experience of a stylized relaxation procedure and were studied prior to learning TM. These two groups will be referred to as the TM and R groups, respectively.

As far as possible, subjects were studied at the same time between 10:30 and 12:00 A.M., while comfortably seated in a dimly lit room. To minimize the effect of testing and interaction of testing with practice, an experimental design similar to that recommended by Campbell and Stanley (9) was used in which no pretest is employed. Subjects of each of the two groups were studied on two occasions, approximately 1 wk apart, each subject serving as his or her own control. On one occasion (treatment, T), subjects of the TM group were asked to close their eyes and practice TM for 45 min followed by an eyes-open recovery period of 30 min; analogously, subjects of the R group were asked to close their eyes and simply rest on the treatment occasion for 45 min followed by an eyes-open 30-min recovery period. These treatment observations will be referred to subsequently as either TMT for the TM group or RT for the R group. On the other (control, C) occasion, subjects of each of the two groups were asked to read a "relaxing" work of their own choice for 45 min followed by a recovery period of 30 min without reading. These control observations will be referred to subsequently as either TMC for the TM group or RC for the R group. The sequence of treatment and reading periods was randomized; in this design, significant departure from constancy of physiological values during treatment and posttreatment recovery periods was measured and contrasted with the trends during the parallel reading and postreading occasion. All subjects were told that it was acceptable to sleep during the practice period if that was their tendency.

After catheters were inserted into a brachial artery and a large antecubital vein of one arm from which blood could easily be drawn without use of a tourniquet, measurements were begun after 2 h to allow physiological changes associated with venipuncture (27) to abate. During this time, subjects were comfortably seated in an enclosed space (6 × 6 × 4 ft) and extension lines for blood drawing and leads for monitoring electrophysiological parameters were attached. Unipolar electroencephalogram, electrooculogram, and electromyogram records were monitored and scored according to standard methods (23) in 14 of the TM subjects and 11 of the R group subjects to determine possible contribution of sleep to metabolic or blood flow changes. A 7-ml sample of arterial and a 7-ml sample of venous blood were taken every 15 min throughout practice and postpractice periods (at times 0, 15, 30, 45, 60, and 75 min) for determination of blood gases and lactate. Arterial and venous blood gases were measured with a Radiometer ABL 1 Blood Gas Laboratory (Radiometer, Copenhagen, Denmark); oxygen saturation was calculated from measured P_{O_2}, P_{CO_2}, and pH by the device. Lactate was determined by a Technicon AutoAnalyzer procedure (14) (Technicon Instrument, Tarrytown, NY). Oxygen content was assumed to be equal to a physically dissolved component (0.003 Pa_{O_2} ml/dl blood) plus the product of measured hemoglobin concentration and calculated oxygen saturation (1.34 × hemoglobin × fractional saturation). As a check on the possibility of a shift in the oxygen-hemoglobin saturation curve, which would render calculated saturations erroneous, oxygen saturation was also measured directly in five subjects (American Optical Oximeter II, NY) of each group and compared with calculated values. Finally, in seven subjects, oxygen content was measured directly by gas chromatography (29) and compared with calculated values.

We chose quadripolar electrical impedance plethysmography (Minnesota Impedance Cardiograph: model 304A, Zoecon, Minneapolis, MN) as the most optimal method for continuous minimally disturbing measurements of relative change of limb blood flow in normal individuals under resting conditions (6, 8, 11, 21, 23, 34). In this technique, electrodes, consisting of 1 mil aluminum bonded to nonallergenic clear plastic tape were placed on the forearm 1, 3, 6, and 8 in. above the wrist. The forearm was comfortably situated at heart level and subjects were asked to move this arm minimally during the course of the experiment. A 1-mA peak-to-peak 100-kHz current was passed through the two outer electrodes and voltage change (due to tissue impedance change) monitored between the two inner electrodes. Quadripolar, rather than bipolar, electrode measurement system was utilized to minimize electrode artifacts (8, 21, 34). To maximize recording accuracy, the electrical impedance signal as well as the electrocardiogram were recorded either online or on a four-channel Tandberg analog tape recorder (Tandberg of America, Armonk, NY) for subsequent analysis on a PDP 11/34 computer system (Digital Equipment, Maynard, MA). Using the R wave of the electrocardiogram as a trigger for beginning the digitization and storage of each impedance pulse, an average electrical impedance waveform was calculated and displayed for each 5-min interval of the experimental period; the average signals obtained were well defined (see Fig. 1). In this way 15 average electrical impedance waveforms, each corresponding to a complete cardiac cycle, were obtained for the experimental period; for each average waveform, pulse amplitude (ΔZ) and late diastolic slope (from 0.4 to 0.9 s) were then ascertained.

Based upon a model of the limb as a homogeneous electrical conductor, a change in volume (ΔV) of the forearm due to each arterial blood pulse is directly proportional to the accompanying change in electrical impedance (ΔZ). Pulsatile blood flow (PBF) is then proportional to heart rate (HR) and ΔV; i.e., $PBF = k \times HR \times \Delta Z$ (11, 23, 34), where k is a function of electrode separation, total electrical impedance, and conductivity of blood. Therefore, the relative pulsatile blood flow

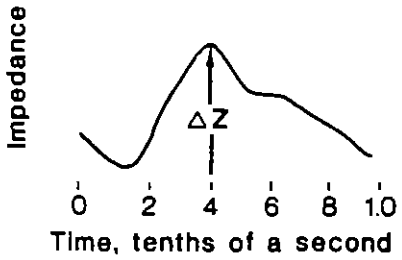

FIG. 1. Typical average electrical impedance change between 2 forearm electrodes during 1 complete cardiac cycle.

between two times can be calculated from

$$\frac{PBF_2}{PBF_1} = \frac{HR_2\Delta Z_2}{HR_1\Delta Z_1} \quad (1)$$

Percent change of relative forearm pulsatile blood is equal to

$$\left(\frac{HR_2\Delta Z_2}{HR_1\Delta Z_1} - 1\right) \times 100 \quad (2)$$

Equation 2 was utilized to calculate change of pulsatile blood flow from initial value (Figs. 4 and 5), where ΔZ_2 was the amplitude of the average impedance pulse measured at each 5-min interval beginning at *time 0* min and ΔZ_1 was the amplitude of the average pulse for the first 5-min interval. HR_2 and HR_1 were the corresponding average heart rates for these same intervals.

An alternative method of calculating limb blood flow change (8) based on the impedance curve was also utilized. This method employs the descending slope in late diastole (from 0.4 to 0.9 s; see Fig. 1); the slope values replace the ΔZ terms in *Eqs. 1* and *2*. On the assumption of zero arterial flow in late diastole, volume changes of the limb during this period are solely due to the difference between inflow and outflow (8). For normal individuals under resting conditions, this assumption is valid, and high correlation of this method of impedance determination of flow with integrated ultrasonic Doppler flow velocity waveform has been demonstrated (8).

Using the Fick principle (14), relative forearm oxygen consumption can be calculated from

$$(\dot{V}O_2) = (CA_{O_2} - Cv_{O_2}) \times PBF \quad (3)$$

Therefore, percent change of forearm oxygen consumption between any two times is

$$\left[\frac{(CA_{O_2} - Cv_{O_2})_2 \times PBF_2}{(CA_{O_2} - Cv_{O_2})_1 \times PBF_1} - 1\right] \times 100 \quad (4)$$

Equation 4 was utilized to calculate percent change of oxygen consumption from initial value (Figs. 2 and 3), where $(CA_{O_2} - Cv_{O_2})_2$, is the arteriovenous difference of oxygen content measured at 0, 15, 30, 45, 60, and 75 min and $(CA_{O_2} - Cv_{O_2})_1$ is that measured at *0 min*, while PBF_2/PBF_1 was evaluated from *Eq. 1* at these same times. *Equation 4* was also employed to calculate percent change of forearm lactate generation rate (Tables 1–4) by substitution of arteriovenous differences of lactate for the oxygen difference terms.

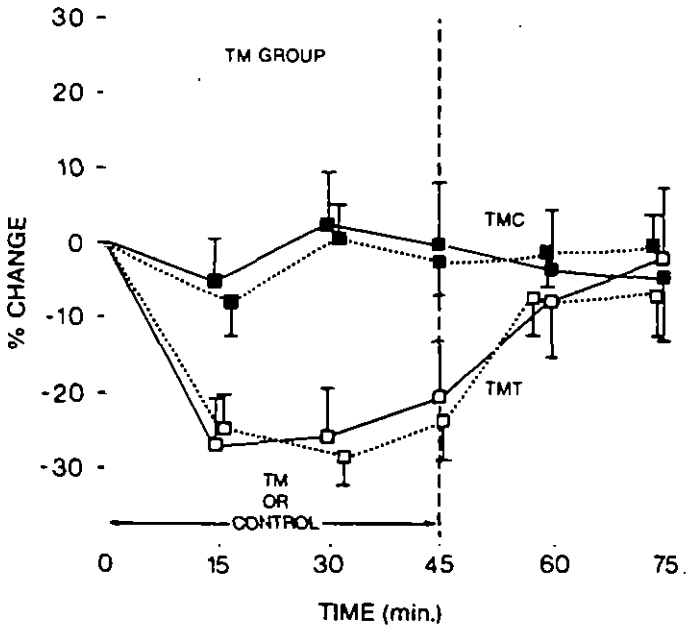

FIG. 2. Mean percent change (±SE) of forearm oxygen consumption (——) (*Eq. 4*) and of arteriovenous oxygen content difference (– – –) during and after TM (□) (TMT) or reading control (■) (TMC).

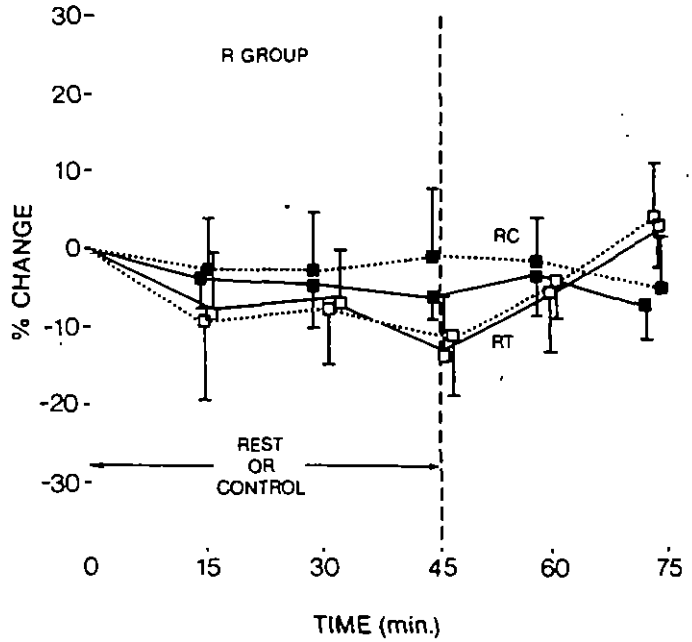

FIG. 3. Mean percent change (±SE) of forearm oxygen consumption (——) (*Eq. 4*) and of arteriovenous oxygen content difference (– – –) during and after eyes-closed unstylized rest (□) (RT) or reading control (■) (RC).

For analysis of change of relative forearm arteriovenous oxygen content difference, oxygen consumption, lactate production, and relative blood flow, curvilinear regression analysis of variation over time with test of significance of the coefficients was used in statistical treatment of the data (30). Comparison of trends between treatment and control reading occasions was accomplished by analysis of variance with time and treatment as classification variables.

TABLE 1. *Mean values, Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, lactate content, and % change of lactate generation during and after TM in TM group (TMT)*

	Time, min					
	0	15	30	45	60	75
Pa_{O_2}, mmHg	102.2 ± 1.2	101.7 ± 1.3	102.4 ± 2.4	102.5 ± 1.4	104.6 ± 1.6	104.5 ± 1.5
Sa_{O_2}, %	96.6 ± 0.2	96.4 ± 0.2	96.7 ± 0.2	96.9 ± 0.3	96.9 ± 0.2	96.9 ± 0.2
Lactate (arterial)*, mg/100 ml	5.6 ± 0.4	4.8 ± 0.6	4.2 ± 0.7	4.0 ± 0.6	6.0 ± 0.7	5.7 ± 1.0
Pv_{O_2}, mmHg	40.1 ± 1.9	47.9 ± 2.3	46.5 ± 1.9	43.3 ± 1.7	41.5 ± 3.4	38.4 ± 2.7
Sv_{O_2}*, %	70.3 ± 2.5	77.8 ± 2.0	77.4 ± 1.7	75.3 ± 1.8	71.4 ± 2.9	66.0 ± 3.5
Lactate (venous)*, mg/100 ml	6.2 ± 0.4	6.6 ± 0.5	5.0 ± 0.7	5.0 ± 0.6	6.2 ± 0.3	5.8 ± 0.7
Lactate generation change, %		5.0 ± 2.4	−0.6 ± 2.3	6.4 ± 3.9	0.1 ± 1.4	0.4 ± 1.2

Values are means ± SE. Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, arterial and venous O_2 tension and saturation; TM, transcendental meditation. * Significant trends.

TABLE 2. *Mean values, Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, lactate content, and % change of lactate generation during and after reading control in TM group (TMC)*

	Time, min					
	0	15	30	45	60	75
Pa_{O_2}, mmHg	100.6 ± 1.16	104.4 ± 1.20	101.7 ± 1.07	99.9 ± 1.00	102.4 ± 1.11	100.1 ± 1.20
Sa_{O_2}, %	95.8 ± 0.2	96.9 ± 0.1	96.4 ± 0.2	95.2 ± 0.2	96.7 ± 0.2	95.4 ± 0.1
Lactate (arterial), mg/100 ml	6.1 ± 0.7	5.4 ± 0.9	6.1 ± 0.8	5.7 ± 0.7	6.1 ± 0.2	4.7 ± 0.7
Pv_{O_2}, mmHg	40.2 ± 2.0	42.6 ± 1.8	41.5 ± 2.4	43.6 ± 2.9	41.5 ± 1.7	41.2 ± 2.0
Sv_{O_2},%	70.4 ± 2.6	72.8 ± 1.9	71.4 ± 2.5	71.4 ± 2.7	73.1 ± 2.0	71.2 ± 2.0
Lactate (venous), mg/100 ml	7.5 ± 0.7	7.8 ± 0.5	8.5 ± 0.5	6.9 ± 1.3	6.9 ± 0.7	4.9 ± 0.9
Lactate generation change, %		1.4 ± 1.6	3.6 ± 1.4	−4.2 ± 1.9	3.6 ± 1.5	0.1 ± 0.9

Values are means ± SE. Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, arterial and venous O_2 tension and saturation; TM, transcendental meditation.

RESULTS

Tables 1–4 show mean values of whole blood arterial and venous oxygen tension, oxygen saturation, lactate, and percent change of lactate generation during the experiment during TMT, RT, TMC, and RC. The major changes noted are significant increases of venous oxygen tension and saturation and significant declines of arterial and venous lactate during TMT (Table 1); the lactate decrease has also been reported previously (19, 31). Figures 2 and 3 show percent change of forearm oxygen consumption and of arteriovenous O_2 content difference from first determination (time 0 min) during TMT, RT, TMC, and RC. Initial mean values (±SE) of arteriovenous difference of oxygen content were 6.86 ± 0.81 for TMT and 5.23 ± 1.20 ml/dl blood for RT, at *time 0* min. Forearm oxygen consumption declined significantly during TMT (28%) and tended to decline during RT (11%), followed by recovery afterward; the trend during TMT, differed significantly from the trend during TMC, which was not associated with significant change. The trends of forearm O_2 consumption (*Eq. 3*) closely paralleled the trends of arteriovenous O_2 difference.

The mean difference (±SE) between measured and calculated arterial and venous saturation values was 0.5 ± 0.6% with a correlation coefficient of 0.996. Mean difference (±SE) between calculated and measured oxygen content values was 0.1 ± 0.6 ml/dl blood with a correlation of 0.945.

Figures 4 and 5 show percent change of impedance determination of relative limb blood flow calculated from *Eq. 2* utilizing mean pulse amplitude. A small increase of flow during TMT was noted, but this trend was not

TABLE 3. *Mean values, Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, lactate content, and % change of lactate generation during and after unstylized eyes-closed rest in R group (RT)*

	Time, min					
	0	15	30	45	60	75
Pa_{O_2}, mmHg	101.5 ± 1.9	99.7 ± 2.0	101.1 ± 2.2	97.7 ± 1.3	100.1 ± 2.2	98.8 ± 1.1
Sa_{O_2}, %	97.5 ± 0.6	96.8 ± 1.0	97.2 ± 0.7	96.4 ± 0.4	98.5 ± 0.8	98.5 ± 0.9
Lactate (arterial), mg/100 ml	6.4 ± 1.0	5.9 ± 0.8	5.5 ± 0.7	5.4 ± 0.8	6.0 ± 0.7	5.8 ± 0.4
Pv_{O_2}, mmHg	42.8 ± 2.9	45.6 ± 7.0	43.3 ± 5.4	44.4 ± 1.9	42.5 ± 3.0	36.5 ± 3.7
Sv_{O_2}, %	65.3 ± 2.1	65.0 ± 2.1	62.6 ± 2.9	65.4 ± 1.4	64.4 ± 1.0	64.0 ± 0.2
Lactate (venous), mg/100 ml	7.2 ± 0.9	6.8 ± 1.2	6.9 ± 0.6	6.2 ± 0.7	7.1 ± 1.1	6.5 ± 0.7
Lactate generation change, %		4.2 ± 2.7	−0.9 ± 1.8	1.1 ± 1.0	3.2 ± 2.7	−4.6 ± 2.1

Values are means ± SE. Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, arterial and venous O_2 tension and saturation.

TABLE 4. *Mean values, Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, lactate content, and % change of lactate generation during and after reading control in R group (RC)*

	Time, min					
	0	15	30	45	60	75
Pa_{O_2}, mmHg	100.6 ± 2.1	99.7 ± 1.4	98.6 ± 1.8	99.8 ± 1.7	101.2 ± 2.8	100.4 ± 1.7
Sa_{O_2}, %	95.5 ± 0.4	95.2 ± 0.3	94.7 ± 0.5	95.3 ± 0.4	96.0 ± 0.5	100.8 ± 0.7
Lactate (arterial), mg/100 ml	6.9 ± 1.8	6.2 ± 1.7	6.4 ± 1.6	6.2 ± 1.4	5.8 ± 0.8	5.6 ± 1.7
Pv_{O_2}, mmHg	43.1 ± 2.8	44.6 ± 3.4	45.2 ± 2.8	42.5 ± 2.1	41.6 ± 4.2	42.1 ± 3.9
Sv_{O_2},%	74.4 ± 5.8	75.2 ± 4.9	76.2 ± 3.8	76.1 ± 5.9	72.4 ± 4.8	73.2 ± 5.4
Lactate (venous), mg/100 ml	8.2 ± 2.6	7.9 ± 2.3	7.6 ± 2.1	6.8 ± 2.5	7.8 ± 0.8	6.7 ± 1.8
Lactate generation change, %		0.8 ± 2.4	−4.2 ± 2.2	0.9 ± 1.6	2.6 ± 1.8	1.1 ± 4.0

Values are means ± SE. Pa_{O_2}, Pv_{O_2}, Sa_{O_2}, Sv_{O_2}, arterial and venous O_2 tension and saturation.

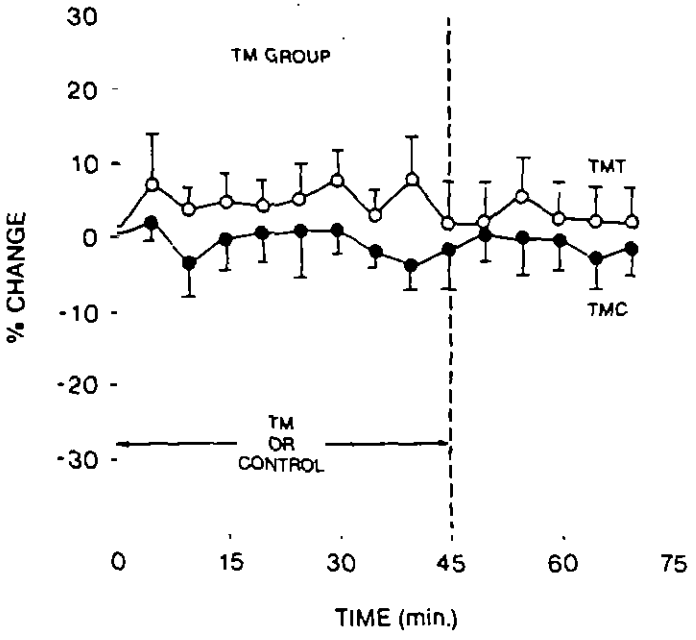

FIG. 4. Mean percent change of pulsatile forearm blood flow (±SE) during and after TM (○) (TMT) or reading control (●) (TMC). Results calculated from *Eq.* 2.

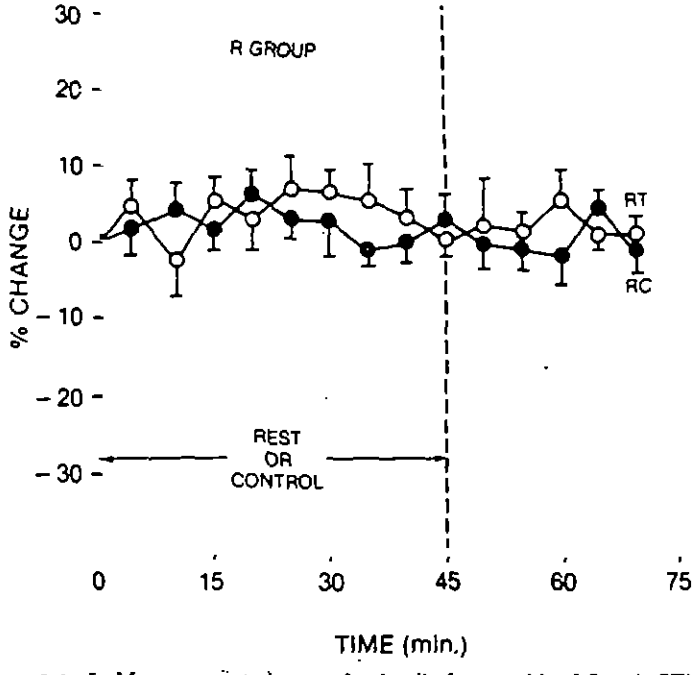

FIG. 5. Mean percent change of pulsatile forearm blood flow (±SE) during and after eyes-closed unstylized rest (○) (RT) or reading control (●) (RC). Results calculated from *Eq.* 2.

significantly different from the trend of blood flow change during the TMC occasion, which was not, itself, associated with significant change (Fig. 4). No change of flow occurred in the rest group during either treatment or control studies (Fig. 5). There was no significant difference between these blood flow results and those calculated by substitution of descending slope values in place of pulse amplitude values in *Eq.* 2.

No significant change occurred in lactate generation by forearm during TM, rest, or control (Tables 1–4). On the average, 90% of the TM period was spent in wakefulness with the remainder consisting of stage I sleep, while ordinary rest in the R group consisted of 86% wakefulness with the remaining period spent in stage I sleep. No correlation existed between percent total sleep time and forearm oxygen consumption or forearm blood flow changes in either group.

DISCUSSION

In this study, we have found that the hypometabolic state associated with the stylized practice of TM is accompanied by acute decrease of forearm oxygen consumption and no significant change of glycolytic metabolism as manifested by lactate uptake and release. Because blood flow changed minimally, the observed decline of oxygen consumption was due almost entirely to decreased rates of tissue oxygen extraction. The initial mean values of arteriovenous oxygen difference for TM and R groups, 6.86 and 5.23 ml/dl, respectively, are within the range of previously reported values (4.95–7.30 ml/dl) attributed to oxygen extraction by resting forelimb muscle (2, 7). One limitation of the accuracy of pulsatile blood flow change as a measure of change of total limb blood flow is the possible existence of a significant steady-state component of flow into the limb. However, in normal individuals under resting conditions such as existed during this experiment, and with the limb at heart level, a linear relationship exists between pulsatile and total limb blood flow (10); and high correlation between pulsatile electrical impedance blood flow determination and direct measurement of total flow by electromagnetic flowmeter has been demonstrated (11).

This study suggests that very little of the previously observed (19) increase of nonrenal nonhepatic blood flow change during TM or ordinary rest could be accounted for by change of muscle and/or skin blood flow and is therefore consistent with preliminary indication of TM-induced increased cerebral blood flow (18). This conclusion assumes that forearm blood flow changes are representative of skin and skeletal muscle blood flow changes in other parts of the body, an assumption supported by comparisons between blood flow changes in arms and calf (5, 32) and comparison between systemic and forearm circulatory changes (1).

The present study does not establish the mechanism by which decreased forelimb oxygen extraction occurs. Electromyographic evidence, however, indicates decreased muscle tone during TM (4), which supports contribution of decreased muscle metabolism in the decline of limb metabolism. Another possibility to explain narrowing of arteriovenous forearm oxygen difference during TM is redistribution of forearm blood flow to less active metabolic sites such as skin. However, estimates of skin blood flow under room temperature conditions, such as apply here, indicate that the skin accounts for only about 15% of forearm blood flow at rest and has about 15% of the metabolic rate of muscle (2, 7). Therefore, skin blood flow would be required to more than triple, and an associated extreme decline in muscle blood flow would be required to account for the observed increase of venous oxygen concentration. Normally, skin

flow is under sympathetic vasoconstrictor control and forearm skin blood flow is little increased by local sympathectomy (28); hence, such a large increase of forearm skin blood flow due to a behavioral intervention seems unlikely. Recently, Benson and colleagues reported that, during an advanced Tibetan Buddhist meditational technique, known as g Tum-mo yoga, practiced for the alteration of body temperature in cool-cold ambient temperatures, finger and forearm temperatures can rise several degrees, possibly by the mechanism of vasodilitation (6). However, vasodilitation of skin forearm and fingers during the procedure of TM (31), practiced in comfortable room temperature conditions, seems unlikely, because galvanic skin resistance (GSR) measured on the hand increased markedly (31), inconsistent with major increase of skin temperature (22). Additionally, Kanellakos and Lukas (20) reported no consistent change of hand temperature during TM practice, and Bagchi and Wenger (3) noted a 1°C decline of forearm temperature during a yogic meditation technique. Therefore, muscle metabolism apparently declines despite little change of muscle blood flow. This study is therefore consistent with the hypothesis of significant contribution of muscle relaxation to the overall decline of metabolic rate in TM (12).

The finding of 10% stage 1 sleep during TM and 14% stage I sleep during ordinary rest plus the lack of correlation between sleep time and forearm oxygen consumption do not support a significant contribution of sleep to the physiological effects of TM, in agreement with previous findings in this laboratory (16, 19).

The observed decrease of arteriovenous oxygen difference and, therefore, oxygen consumption (*Eq. 3*) might also only be apparent, i.e., due to a shift of the oxygen-hemoglobin saturation curve to the right, so that oxygen was released with greater ease. However, such disturbance of normal P_{O_2} saturation relationship seems unlikely, because measured and calculated arterial and venous saturation and content values were almost identical.

Lack of change of forearm lactate uptake-release diminishes the likelihood of skin or muscle contribution to the decline of serum lactate observed during TM [Table 3; (19, 31)]. The data therefore support decreased red cell lactate generation as probable contributor to the decline of blood lactate concentration based on a related study indicating marked decline of erythrocyte glycolytic rate during this behavior (17) and the fact that red cell glycolysis is a primary contributor to total blood lactate content in resting humans (14). The observation that there is concomitant decrease of red cell metabolism during TM raises the possibility of modulation of the metabolic changes by circulating factor(s).

Of considerable interest is the clear dissociation of limb blood flow and metabolic changes (Figs. 2–5). These data, and the previously observed small increase of cardiac output (19) during this state of decreased activation, suggest limitation of the hypothesis of obligatory coupling between systemic and/or regional cardiovascular and metabolic function (24) over the complete range of physiological activation associated with changes of behavioral origin. The data also indicate small contribution of altered muscle blood flow to the previous finding of increased nonrenal nonhepatic blood flow in this state (15).

The authors acknowledge the generous assistance of Drs. William D. Davies, Torrance Memorial Hospital, and William G. Kubicek, Dept. of Physical Medicine and Rehabilitation, The University of Minnesota, in this research. We also thank the TM teachers of the Orange County World Plan Center whose participation as subjects made this study possible.

We also acknowledge the financial support of the National Institute of Mental Health Grant MH-29791-02; National Heart, Lung, and Blood Institute Grant HL-27894-01; and The John and Catherine MacArthur Foundation.

This research was presented in part at the Spring Meeting of the American Physiological Society, Anaheim, CA. April, 1980.

Received 17 May 1982; accepted in final form 13 December 1982.

REFERENCES

1. ABRAMSON, D. I., AND E. B. FARRIS. Responses of blood vessels in the resting hand and forearm to various stimuli. *Am. Heart J.* 19: 541–553, 1940.
2. ANDRES, R., G. CADER, AND K. L. ZIERLER. The quantitatively minor role of carbohydrate in the oxidative metabolism by skeletal muscle in intact man in the basal state. Measurements of oxygen and glucose uptake and carbon dioxide and lactate production in the forearm. *J. Clin. Invest.* 35:671–682, 1956.
3. BAGCHI, B. K., AND M. A. WENGER. Electrophysiologic correlates of some yogic exercises. *Electroencephalogr. Clin. Neurophysiol. Suppl.* 17: 132–149, 1957.
4. BANQUET, J. P. The spectral analysis of the EEG during transcendental meditation. *Electroencephalogr. Clin. Neurophysiol.* 35: 142–149, 1973.
5. BARCROFT, H. Blood flow and metabolism in skeletal muscle. In: *Circulation in Skeletal Muscle*, edited by O. Hudlicka. Oxford, UK: Pergamon, 1968, p. 121–187.
6. BENSON, H., J. W. LEHMANN, M. S. MALHOTEA, R. F. GOLDMAN, J. HOPKINS, AND M. D. EPSTEIN. Body temperature changes during practice of g Tum-mo yoga. *Nature London* 295: 234–6, 1982.
7. BROD, J., Z. HEIL, AND M. ULRYCH. Metabolic changes in the forearm muscle and skin during emotional and muscular vasodilation. *Clin. Sci.* 25: 1–19, 1963.
8. BROWN, B. H., W. I. J. PRYCE, AND D. BAUMER. Impedance plethysmography: can it measure changes in limb blood flow? *Med. Biol. Eng.* 13: 674–682, 1975.
9. CAMPBELL, D. T., AND J. C. STANLEY. *Experimental and Quasi-Experimental Designs for Research.* Chicago, IL: Rand McNally, 1968.
10. COOK, M. R. Psychophysiology of peripheral vascular changes. In: *Cardiovascular Psychophysiology*, edited by P. A. Obrist, A. H. Black, J. Brenner, and L. DiCara. Chicago, IL: Aldine, 1974, p. 60–85.
11. COUCH, N. P., J. M. VANDEWATER, AND J. R. DMOCHOWSKI. Non-invasive measurement of peripheral arterial flow. *Arch. Surg.* 102: 435–439, 1971.
12. FENWICK, P. B. C., S. DONALDSON, L. GILLIS, AND J. BUSHMAN. Metabolic and EEG changes during transcendental meditation: an explanation. *Biol. Psychol.* 5: 101–118, 1977.
13. FOLKOW, B., AND E. NEIL. *Circulation.* London: Oxford Univ. Press, 1971.
14. HOACHELLA, N. J., AND S. WEINHOUSE. Automated lactic acid determination in serum. *Anal. Biochem.* 10: 304–312, 1965.
15. HOFFMAN, J. W., H. BENSON, P. A. ARNS, G. L. STAINBROOK, L. LANDSBERG, J. B. YOUNG, AND A. GILL. Reduced sympathetic activity associated with the relaxation response. *Science* 215: 190–192, 1982.
16. JEVNING, R., A. F. WILSON, AND J. M. DAVIDSON. Adrenocortical activity during meditation. *Horm. Behav.* 10: 54–68, 1978.

17. JEVNING, R., A. F. WILSON, AND J. P. O'HALLORAN. Behavioral modulation of red cell glycolysis. *Am. J. Physiol. (Cell Physiol.)*. In press.
18. JEVNING, R., A. F. WILSON, AND J. P. O'HALLORAN. Behavioral increase of cerebral blood flow (Abstract). *Physiologist* 21(4): 60, 1968.
19. JEVNING, R., A. F. WILSON, W. R. SMITH, AND M. E. MORTON. Redistribution of blood flow in acute hypometabolic behavior. *Am. J. Physiol.* 235 (*Regulatory Integrative Comp. Physiol.* 4): R89–R92, 1978.
20. KANELLAKOS, D. P., AND J. S. LUKAS. *The Psychobiology of Transcendental Meditation: A Literature Review*. Menlo Park, CA: Benjamin, 1974.
21. KUBICEK, W. G., R. P. PATTERSON, AND R. C. LILLEHEI. Impedance cardiography as a non-invasive method to monitor cardiac function. *J. Am. Assoc. Adv. Med. Instrum.* 4: 79–84, 1970.
22. MAULSBY, R. C., AND R. EDELBERG. The interrelationship between the galvanic skin response, basal resistance, and temperature. *J. Comp. Physiol. Psychol.* 53: 475–479, 1960.
23. NYBOER, J. *Electrical Impedance Plethysmography*. Springfield, IL: Thomas, 1970.
24. OBRIST, P. A., J. E. LAWLER, AND C. J. GAEBELEIN. A psychobiological perspective on the cardiovascular system. In: *Advances in Limbic and Autonomic Nervous System Research*, edited by L. DiCara. New York: Plenum, 1973, p. 311–334.
25. PAGANO, R. R., R. M. ROSE, R. M. STIVERS, AND S. WARRENBURG. Sleep in transcendental meditation. *Science* 191: 309–310, 1976.
26. RECHTSHAFFEN, A., AND A. KALES. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Los Angeles, CA: Brain Information Service/Brain Research Institute, 1968. (UCLA Natl. Inst. Health Publ. 204)
27. ROUSER, G., AND B. JELINEK. Free amino acids in the blood of man and animals. I. Method of study and the effects of venipuncture and food intake on blood free amino acid. In: *Amino Acid Pools, Distribution, Formation and Function*. New York: Elsevier, 1961, p. 350–372.
28. ROWELL, L. B. The cutaneous circulation. In: *Physiology and Biophysics*, edited by T. C. Ruch and H. D. Patton. Philadelphia, PA: Saunders, 1974, p. 185–199.
29. SHEPHERD, J. C., J. C. SUTHERLAND, AND A. F. WILSON. Continuous spectrophotometric measurement of arteriovenous O_2 difference. *J. Appl. Physiol.* 39:152–155, 1975.
30. SNEDECOR, G. W., AND W. G. COCHRAN. *Statistical Methods*. Ames, IA: Iowa State Univ. Press, 1973, p. 248–302.
31. WALLACE, R. K., H. BENSON, AND A. F. WILSON. A wakeful hypometabolic state. *Am. J. Physiol.* 221: 795–799, 1971.
32. WILKINS, R. W., AND L. W. EICHNA. Blood flow in forearm and calf. *Bull. John Hopkins Hosp.* 68: 425–428, 1941.
33. WILLIAMS, R. B., T. E. BITTKER, M. S. BUCHSBAUM, AND L. C. WYNNE. Cardiovascular and neurophysiologic correlates of sensory intake and rejaection. I. Effect of cognitive tasks (Abstract). *Psychophysiology* 12: 427, 1975.
34. YABLONSKI, M. R., J. M. VAN DE WATER, B. E. MOUNT, E. D. LASKA, AND R. B. INDECH. Calibrated impedance plethysmograph. *Am. J. Physiol.* 239 (*Heart Circ. Physiol.* 8): H283–H288, 1980.