

The Effects of Graduated Exercise at the Piano on the Pianist's Cardiac Output, Forearm Blood Flow, Heart Rate, and Blood Pressure

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Nearly every musician has encountered physiologic stumbling blocks in practice and performance situations. A few virtuosi have such tremendous gifts of physical ease and instinctive adjustment to their tasks that this issue is, for them, minor. Most musicians, however, are far too familiar with the frustration of fruitless practice time and marred performances which result from the inability to circumvent some physical difficulty that stands in the way of successful achievement of a given musical task. It seems likely that certain cases of suboptimal practice and performance-related injury might stem from a misunderstanding of human physiology and the physiologic aspects of playing a musical instrument.

The purpose of this investigation was to explore the cardiovascular responses of pianists to varying levels of physiological stress during piano playing. The parameters considered were cardiac output, forearm blood flow, heart rate, and blood pressure.⁶ Cardiac output, the volume of blood pumped by the heart in one minute, is the volume of blood pumped during each contraction multiplied by the heart rate.

Methods

Three male and twelve female subjects, with a mean age of 30 years (range, 20–43 years) and a mean history of piano practice of 21 years (range, 7–37 years) participated in this study. None was suffering from heart disease or

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circulatory problems, or taking any drugs that could have affected the test results.

Cardiac output, forearm blood flow, and heart rate were monitored on a Model 304 Minnesota Impedance Cardiograph.^{1,2} Blood pressure was measured with a hand-held sphygmomanometer. Subjects performed on an Everett studio upright piano and were monitored with tetrapolar electrode-placement systems on the chest and right arm. (Fig. 1).

Values for cardiac output were recorded in liters (L) per minute. Forearm blood flow values, in milliliters (ml) per minute, were obtained from a 10-cm segment of the right arm which contained the muscles most actively involved in scale playing on the piano—the extensors and flexors of the wrist and fingers. These values were standardized for blood resistivity,^{3*} limb segment volume,⁴ and body surface area.⁴ Cardiac output was converted to cardiac index in L per minute per meters squared (m²) of body surface area.⁵ Forearm blood flow was converted to forearm flow index, expressed as ml per minute per 100 ml of tissue.⁴

In the five minutes preceding exercise, five basal state values for cardiac output, forearm blood flow, heart rate, and blood pressure were recorded. The highest and lowest values for each parameter were discarded and a mean was derived from the remainder.

The piano exercises consisted of four-octave, bilateral scales in sixteenth notes played to a quarter-note beat.

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* Blood resistivity, the electrical resistance of blood, was determined from the hematocrit, the ratio of packed cell volume to the total blood volume.

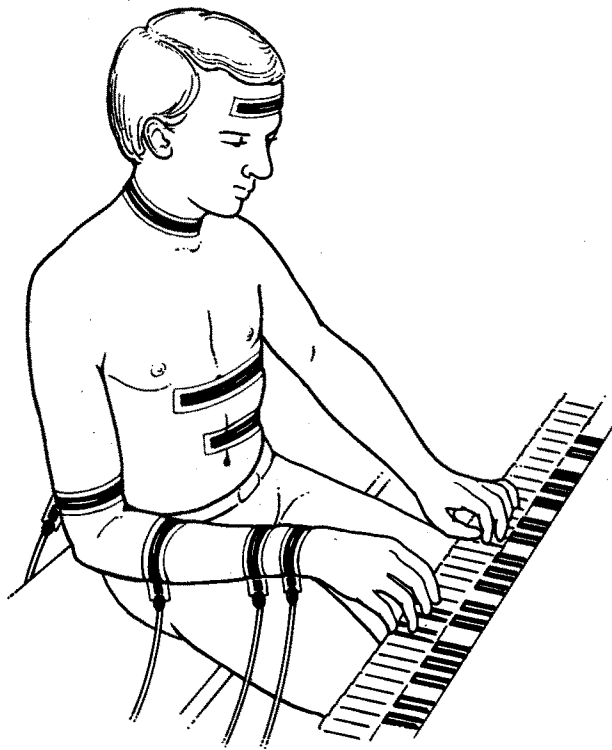


FIGURE 1. The four electrodes on the right arm are for measurement of forearm blood flow. The electrodes on the forehead, neck, chest, and abdomen are for assessment of cardiac output and heart rate.

Exercise was performed in three-minute stages at the following tempi:

Stage I	= 60 beats/minute
Stage II	= 80 beats/minute
Stage III	= 100 beats/minute
Stage IV	= 120 beats/minute
Stage V	= 144 beats/minute
Stage VI	= 160 beats/minute
Stage VII	= 184 beats/minute
Stage VIII	= as fast as possible

Tempi were set and maintained by the use of a Franz electronic metronome.

Cardiac output values were recorded 30 seconds preceding the end of each exercise stage. Blood pressure and forearm blood flow were determined immediately after exercise ceased. Exercise at the next successive stage commenced as soon as all data were recorded and the new exercise tempo was established. Subjects progressed through as many stages of exercise as possible, after which a five-minute recovery period was observed. Values for each parameter were recorded once each minute during this period.

Results

Cardiac Output. Changes in the mean cardiac index from basal state through recovery are shown in Table 1. A decreasing number of participants were capable of participation in higher-stress exercise stages V through VIII.

TABLE 1. Changes in Cardiac Index During Piano Exercise and Recovery

Stage	Number of Participants	Mean Cardiac Index (L/min/m ²)	Increase Over Basal State
Basal state	15	3.1	—
Stage I	15	3.44	8%
Stage II	15	3.65	14%
Stage III	15	3.84	20%
Stage IV	15	4.11	29%
Stage V	14	4.40	38%
Stage VI	10	4.74	48%
Stage VII	5	4.90	60%
Stage VIII	2	4.68	63%
Recovery I	15	3.68	15%
Recovery II	15	3.32	4%
Recovery III	15	3.21	1%
Recovery IV	15	3.15	-1%
Recovery V	15	3.14	-2%

Therefore, trends for percentage change from basal state were determined in each stage using the basal state values of active participants only. The findings show progressive, proportional increases in demand on the heart as exercise increased in tempo, and recovery to basal state within five minutes.

Forearm Blood Flow. Forearm blood flow changes are charted in Table 2, with absolute mean values and percentage change from basal state for the participating subjects in each stage. Flow increased steadily with progressive tempo increases in piano playing. For the two participants who completed stage VIII of exercise, the increase averaged 232% over basal state blood flow volume (201% and 263%, respectively). After only one minute of recovery, a striking decrease in flow was recorded which represented a 192% lesser volume, in relation to basal state, than that which characterized the final exercise stage.

TABLE 2. Changes in Forearm Flow Index During Piano Exercise and Recovery

Stage	Number of Participants	Mean Forearm Flow Index (ml/min/100 ml)	Increase Over Basal State
Basal stage	15	9.8	—
Stage I	15	11.0	12%
Stage II	15	13.4	36%
Stage III	15	16.3	66%
Stage IV	15	19.7	100%
Stage V	14	22.8	136%
Stage VI	10	25.2	160%
Stage VII	5	25.0	185%
Stage VIII	2	26.3	232%
Recovery I	15	13.8	40%
Recovery II	15	13.5	37%
Recovery III	14*	12.2	25%
Recovery IV	15	11.0	12%
Recovery V	14*	9.6	-2%

* Due to mechanical difficulties it was not possible to obtain forearm flow measurements for one subject in Recovery Stages III and V.

For 6 of the 15 subjects increases in peak forearm flow did not occur in the final exercise stage achieved. All of these subjects had difficulty meeting the exercise demands of the final stage, complaining of excessive forearm tension and fatigue and performing in a stumbling manner with lagging tempi in their final exercise stage. In group analysis (Table 2), however, the decreased flows of these six were overshadowed by the increases of the other subjects.

All the pianists reached a ceiling for tempo increase in the piano exercises. It cannot be determined from our data why they were unable to persist after a certain point or why some were not able to persist as long as others. A notable relationship was observed, however, between forearm blood flow responses and complaints of tension and fatigue in the pianists' arms at various levels of stress.

While the mean forearm flow values for the group increased proportionally to increases in exercise intensity in Stages II through VII (Table 2), in each of 11 subjects who exhibited symptoms of arm tension and fatigue, forearm flow leveled out or diminished with fatigue.

Only one pianist was able to "get a second wind" after a bout with arm fatigue. This subject suffered from fatigue in the left arm in Stages V and VI and reached a plateau at 17.7 and 17.9 ml/min/100 ml, respectively, in those stages. The subject revived and suffered no symptoms in Stage VII, when the forearm flow index jumped to 28.7 ml/min/100 ml, a 60% increase over Stage VI. Recurrence of fatigue and tension in Stage VIII was again accompanied by a plateau in the forearm flow index (29.0 ml/min/100).

Heart Rate. Table 3 shows the mean heart rate for each exercise stage as well as the percentage increase in each stage calculated in relation to basal state values of the active participants only. Like cardiac output and forearm blood flow, heart rate increased linearly with exercise intensity for individual participants. Unlike those two parameters (Tables 1 and 2), however, the trend toward increased heart rate was obscured in group analysis. Although increases in heart rate appear to level off between Stages IV and VI and to drop in Stage VIII (Table 3), only two participants were able to tolerate the Stage VIII level of exercise, and both had slightly higher heart rates in Stage VIII (mean, 112 beats/min) than in Stage VII (mean, 109 beats/min). This increase was obscured, however, by the group data for the five participants who completed Stage VII of exercise.

Similarly, the trend toward an increase in heart rate in Stages V and VI was obscured by the group data. This can be attributed to the fact that subjects who persisted less long in the exercise series frequently exhibited incremental increases of greater magnitude, and higher peak heart rates from stage to stage, than subjects who persisted longer.

Table 4 indicates mean heart rates in those stages where subjects achieved peak values. It includes the basal state

TABLE 3. Changes in Heart Rate During Piano Exercise and Recovery

Stage	Number of Participants	Mean Heart Rate (beats/min)	Increase Over Basal State
Basal state	15	81	—
Stage I	15	88	9%
Stage II	15	92	14%
Stage III	15	97	20%
Stage IV	15	105	30%
Stage V	14	105	30%
Stage VI	10	106	39%
Stage VII	5	120	60%
Stage VIII	2	112	47%
Recovery I	15	83	2%
Recovery II	15	80	-1%
Recovery III	15	80	-1%
Recovery IV	15	81	0%
Recovery V	15	82	1%

TABLE 4. Heart Rates in Stages Where Peak Levels Were Achieved

Stage	Number of Subjects with Peak Rate	Mean Basal Rate (beats/min)	Mean Peak Rate (beats/min)	Percent Increase
Stage IV	3	88	127	44%
Stage V	4	84	100	19%
Stage VI	4	80	118	48%
Stage VII	2	73	119	63%
Stage VIII	2	76	112	47%

values, and the percentage increase that the peak rate represents. Subjects who persisted longer in the exercise series recorded lower basal state values than those who ceased exercise sooner. No clear trend is seen, however, for the mean peak values or the percentage increase over basal state which they represent.

Blood Pressure. Table 5 records changes in absolute mean blood pressure and the percentage increase for participating subjects in each stage of exercise. Systolic pressure increased by small increments through the series of exercises, whereas diastolic pressure showed minimal or no change.

Both the mean values and percentage change were considerably affected by the decreasing number of participants in Stages VII and VIII. Whereas five pianists remained in Stage VII, only two remained in Stage VIII. In one of these individuals, the diastolic pressure increased slightly, from 62 mm Hg in Stage VII to 64 mm Hg in Stage VIII; in the other, the diastolic pressure fell from 80 mm Hg in Stage VII to 74 mm Hg in Stage VIII. Thus, the apparent 13% diastolic drop, from 80 mm Hg in Stage VII to 69 mm Hg in Stage VIII (Table 5), was an artifact of the decreasing number of participants. Notably, blood pressure recovered to the basal state level within the five-minute period of observation after the exercise.

Only one pianist was able to "get a second wind" after a bout with arm fatigue.

TABLE 5. Changes in Blood Pressure During Piano Exercise and Recovery

Stage	Number of Participants	Mean (mm Hg)	Increase Over Basal State*
Basal state	15	118/76	— / —
Stage I	15	123/77	4%/ 1%
Stage II	15	126/80	7%/ 5%
Stage III	15	129/80	9%/ 5%
Stage IV	15	133/81	13%/ 7%
Stage V	14	133/80	16%/ 7%
Stage VI	10	135/80	19%/ 7%
Stage VII	5	140/80	24%/10%
Stage VIII	2	141/69	34%/ -3%
Recovery I	15	127/80	8%/ 5%
Recovery II	15	122/79	3%/ 4%
Recovery III	15	121/78	3%/ 3%
Recovery IV	15	119/78	1%/ 3%
Recovery V	15	119/79	1%/ 4%

*Percentages are expressed as systolic pressure change/diastolic pressure change.

Discussion

Increases in cardiac output may be due either to general body stress or increased need in a specific body area. Therefore, it is theoretically possible that the increase in cardiac output in our subjects was due mainly to increased forearm blood flow during exercise. Table 6 shows cardiac output and blood flow to both forearms for the basal state and each exercise stage, and changes from stage to stage for both parameters. The data show that the increases in cardiac output resulting from the exercises were far greater than the additional blood volume required by the forearms to execute the exercises.

It thus appears that strenuous piano playing stresses not only the active muscles in the forearms, but the body in general. The precise causes of the stress can only be surmised. It is likely that a very small portion of the excess blood flow was required in active muscle beds not measured in this experiment, such as the upper arm, shoulder girdle, and back. The percentage of the total excess flow repre-

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sented by these areas would have been minimal, however, since they were not nearly as heavily stressed by the exercise as the forearms. For the same reason, only a very small portion of the excess flow might be attributed to unnecessary muscular tension in other areas of the body.

In this study, the mean basal state cardiac output was 5.34 L/min and the mean peak increase* in cardiac output was 7.82 L/min. The highest peak cardiac output recorded by any subject was 11.4 L/min. In practical terms, cardiac output changes indicate the physiological stress a given activity places upon the heart. It is commonly accepted that cardiac output relates linearly to exercise intensity until maximum cardiac output capability is reached. The average cardiac output for a normal resting adult is 5 L/min and can increase to as high as 20 to 25 L/min with strenuous activity.⁵ Walking may result in levels as high as 7.5 L/min.⁶ It appears from our data that the stress placed on the heart by vigorous piano playing can be at least as great as that produced by a brisk walk.

It is not possible to determine how stress-provoking the testing procedures were or whether the large cardiac output response that we observed would have occurred as a result of strenuous piano playing in a nontested private practice session. However, since for professionals as well as for many amateurs the goal of piano playing is not the private practice session, our testing procedure presumably reflected fairly

*For some subjects cardiac output peaked in the penultimate exercise stage rather than in the final stage, due to fatigue and lagging performance tempo in the final stage. This value for cardiac output is, therefore, based on the peak increase for each subject rather than on the value obtained in the final exercise stage. Note also that raw cardiac output values, rather than the standardized cardiac index values in Table 1, are being used here.

TABLE 6. Mean Cardiac Output and Forearm Flow Increases

Stage	Cardiac Output		Forearm Flow		Excess Cardiac Output Increase [†] (ml/min)
	(ml/min)	Increase Over Preceding Stage (ml/min)	(ml/min)*	Increase Over Preceding Stage (ml/min)	
Basal state	5,340	—	70	—	—
Stage I	5,750	410	81	11	399
Stage II	6,120	370	95	14	356
Stage III	6,440	320	115	20	300
Stage IV	6,870	430	139	24	406
Stage V	7,290	420	152	13	407
Stage VI	7,670	380	167	15	365
Stage VII	8,050	380	163	-4	384
Stage VIII	7,170	-880	163	0	-880

*Values for forearm flow have been doubled to approximate the blood flow to both forearms.

†Values indicate the increase in cardiac output (ml/min) in excess of the increase in forearm flow.

accurately—or even underestimated—what happens to cardiac output under conditions of stress in piano playing.

The forearm flow increases of approximately 30% (Table 2) in each of stages II through VII suggest that, at submaximal levels of stress, increases in forearm blood flow are proportional to increases in exercise intensity.

Basal state and peak heart rates, as well as the magnitude of increase from stage to stage, served as reasonably good indices of our pianists' endurance capabilities, since those who exhibited lower basal state and peak rates and smaller incremental changes were most frequently those who endured longer in the exercise regimen (Table 4). These findings parallel observations of heart rates in trained athletes.⁷ It seems reasonable that those pianists who persisted longer in the exercise series were demonstrating the effects of some physical training. A larger number of subjects would be required to verify this observation and determine whether the heart rate response might be related to general cardiovascular conditioning, piano practice conditioning, or both.

For any physical exercise there is a potential for enhanced exercise performance if significant repetition, or training, occurs. The major factors leading to training improvement are: (1) initial fitness level, (2) intensity of exercise, (3) duration of exercise, (4) frequency of exercise and (5) type of exercise.⁸

One aspect of enhanced exercise performance relates to adaptive changes in the muscles most utilized in a given exercise. A second aspect is a more generalized cardiovascular benefit relating to improved aerobic fitness* for the given exercise activity.⁸

The maximum heart rate for an individual has been established as 220 minus the person's age in years.⁸ As a general rule, aerobic capacity† improves if exercise is of sufficient intensity to increase the heart rate to about 70% of maximum.⁸ When training involves only arm exercise, however, the maximum heart rate required to enhance aerobic capacity is 70% of a value approximately 13 beats per minute lower than the predicted maximum heart rate for age [(220 - age in years) - 13].⁸ In our study seven pianists (age range, 27-46 years) achieved or overshot the 70% target rate. These individuals developed heart rates that represented 69% to 83% of their age-predicted maximum heart rates. Thus, stressful piano playing would appear to provide sufficient exercise intensity to improve aerobic capacity if the duration of the workout were sufficient. The appropriate duration for such exercise would be dependent on factors such as total work done, exercise intensity through

*Aerobic fitness refers to a variety of functional capacities related to oxygen transport and use in the body.⁸

†Aerobic capacity is the level of oxygen consumption at which, in a given individual, an increase in workload no longer produces an increase in oxygen consumption. It is an important point of reference in exercise physiology as it is a stable and highly reproducible characteristic of an individual that serves as a measure of the functional capacity of the cardiovascular system.⁸

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the work period, frequency of exercise and initial fitness level, all of which would vary from pianist to pianist.⁵

A value of 120/80 mm Hg is accepted as an average basal state blood pressure for young adulthood.⁹ Sheffield reported that the normal systolic pressure after near-maximal exercise was approximately 120 to 200 mm Hg, with a mean of 160 mm Hg, and that the diastolic pressure change was unlikely to exceed 10 mm Hg.⁹ In fact, diastolic pressures in younger individuals may fall slightly as exercise intensity increases.¹⁰ Our findings are in line with these observations (Table 5). In addition, they indicate that piano exercise can present a work load great enough to cause a slight drop in diastolic pressure (Table 5).

The major limitation of this study was the small number of participants. Data could thus not be subjected to statistical analysis. Larger subject numbers would allow, in particular, analysis of the effects of piano practice conditioning and general cardiovascular conditioning on the outcome measures we examined so that training effect variables could be considered.

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References

1. Kubicek WG, Patterson RP, Witsoe DA, et al: Impedance cardiography as a noninvasive means to monitor cardiac function. *J Assoc Adv Med Instrum* 4:79-84, 1970.
2. Wheeler HB, Penney BC: Impedance plethysmography: theoretical and experimental basis. In Bernstein EF (ed): *Noninvasive Diagnostic Techniques in Vascular Disease*. St. Louis, C.V. Mosby Co., 1982, pp 104-116.
3. Geddes LA, Sadler C: The specific resistance of blood at body temperature. *Med Biol Eng* 11:336-339, 1973.
4. Parr S: The effects of graduated exercise at the piano on the pianist's cardiac output, forearm blood flow, heart rate, and blood pressure. Doctoral Dissertation, Ball State University, 1985.
5. Guyton AC: Cardiac output, venous return, and their regulation. In: *Textbook of Medical Physiology*, 7th ed. Philadelphia, W.B. Saunders, 1986, pp 272-286.
6. Guyton AC: Cardiac output, venous pressure, cardiac failure, and shock. In: *Function of the Human Body*, 4th ed. Philadelphia, W.B. Saunders, 1974, pp 140-151.
7. Milnor WR: Normal circulatory function. In Mountcastle VB (ed): *Medical Physiology*, 14th ed. St. Louis, C.V. Mosby Co., 1979, pp 1033-1046.
8. McArdle WD, Katch FI, Katch VL: Training for anaerobic and aerobic power. In: *Exercise Physiology*. Philadelphia, Lea and Febiger, 1986, pp 347-370.
9. Sheffield LT: Exercise stress testing. *Heart Disease*. In Braunwald E (ed): *A Textbook of Cardiovascular Medicine*. Philadelphia, W.B. Saunders, 1980, pp 258-278.
10. Blomquist CG: Clinical exercise physiology. In Wenger NK (ed): *Rehabilitation of the Coronary Patient*. New York, John Wiley and Sons, 1978, pp 133-148.