Hemodynamic and Metabolic Responses to Upright Exercise in Patients with Congestive Heart Failure*

TREATMENT WITH NITROGLYCERIN OINTMENT

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Although a vasodilator drug might favorably alter hemodynamics in resting, supine patients with heart failure, it must be determined if the drug has any unfavorable as well as favorable effects under extremes of real-life stress situations normally encountered by ambulatory patients, especially upright exercise. An adverse response might occur with the addition of pharmacologic vasodilation to the metabolic arteriolar dilation accompanying upright exercise. In order to evaluate these factors in a controlled environment, seven NYHA class 2-3 patients with congestive heart failure without angina pectoris or stenotic valvular lesions performed upright bicycle exercise pre- and postadministration of a maximally-tolerated dose of 2 percent nitroglycerin ointment, determined previously by titration. Nitroglycerin therapy resulted in slightly improved cardiac performance at two given submaximal workloads. Also noted was, at a higher workload, a decreased ratio of arteriovenous oxygen difference to cardiac index suggestive of increased circulatory reserve. Pulmonary capillary wedge pressure, total body oxygen consumption, plasma catecholamines, and blood lactates did not show a statistically significant change after use of nitroglycerin ointment, nor were symptoms of fatigue or dyspnea improved during exercise. It is concluded that nitroglycerin ointment does not remarkably improve hemodynamics or alter nutritional blood flow to exercising muscle at a submaximal upright workload. From our data and that of others, it is concluded that nitroglycerin ointment can be given safely to heart failure patients to improve resting supine hemodynamics without adversely affecting upright exercise performance or hemodynamics.

Administration of vasodilator drugs has proved useful in the therapy of many patients with congestive heart failure.1-10 Depending upon the drug used, improvement in cardiac performance in resting patients has been manifested by lowering left ventricular filling pressure and/or an increase in cardiac index.10 The former effect seems to predominate when nitrates are given and it has been suggested that they have a predominately venodilator action. However, nitrates can also dilate resistance vessels, especially when basal systemic vascular resistance is elevated.8 On the basis of the known effects of nitrates on the circulation and the known peripheral vascular abnormalities accompanying congestive heart failure,10-11 it is difficult to predict how these drugs will alter the distribution of the cardiac output during exercise.

In a given patient at a particular time, a number of factors can contribute to the increased systemic arterial resistance noted in congestive heart failure; examples of these are an intrinsic “vascular stiffness” (related to increased sodium content of blood vessels), elevated humoral vasoconstrictors (catecholamines and angiotensin), and increased neurogenic vasomotor tone.10-11 The result of this increased arteriolar tone is that in many congestive heart failure patients, the peripheral resistance vessels do not dilate normally in response to exercise,12-13 skeletal muscle oxygen consumption fails to increase normally, and there is a greater shift to anaerobic metabolism. The resulting reduction in oxygen content of muscles stimulates somatic nerve endings, the afferent limb of the reflex arc that results in excessive sympathetic efferent discharge.11 If administration of a vasodilator drug increased nutritional blood flow to exercising muscle groups, there would be less of a shift to anaerobic metabolism, lactate production would be lower, and oxygen consumption for any given workload would increase. With an improvement in aerobic metabolism.
and less skeletal muscle ischemia, the sympathoadrenal response to exercise might be lessened.\textsuperscript{10}

On the other hand, a vasodilator might worsen performance during exercise. A venodilator, by reducing venous return, could lead to a fall in cardiac output. Similarly, if an arteriolar dilating agent relaxed constricting circulation to which blood flow was being sacrificed to preserve arterial perfusion pressure, exercise syncope might result.\textsuperscript{10}

In the present study, we investigated whether nitroglycerin ointment (TNG-O) produced a favorable or an unfavorable response when patients with moderately severe congestive heart failure underwent upright bicycle exercise at a submaximal workload. Criteria for a favorable response would include improved hemodynamics (lower ventricular filling pressure and higher cardiac output) and an augmented delivery of oxygen to exercising muscles (increased systemic oxygen consumption, decreased lactate production, and reduced plasma catecholamine concentration) with preservation of systemic arterial pressure. The converse would be considered unfavorable responses.

**MATERIALS AND METHODS**

Seven patients (two women, five men) from 26 to 59 years old (mean 46 years) were studied. By description of their symptoms, four patients were judged to be in New York Heart Association functional class 3, and three in class 2.\textsuperscript{14} The cause of heart failure was mitral regurgitation in two subjects, mixed mitral and aortic regurgitation in one patient, isolated aortic regurgitation in one, and three individuals had prior transmural myocardial infarction (two anterior, one inferior). Two patients underwent open heart surgery and prosthetic valve replacement subsequently. Each individual complained of exercise intolerance, but none had angina pectoris of ischemic ST segment changes. Five of the patients had cardiomegaly on their plain chest x-ray films prior to study. One patient, although not demonstrating cardiomegaly, had previously undergone left ventriculography which demonstrated an ejection fraction of 0.33. Each patient underwent pretesting on the bicycle ergometer at least 24 hours prior to the final study in order to obtain an estimate of maximal work capacity, as well as to gain familiarization with the equipment. The pretest exercise was begun at 25 watts and increased by 25-watt increments at three-minute intervals until maximal effort was obtained. Maximal oxygen consumption was estimated from the workload at the conclusion of this exercise,\textsuperscript{15} and workloads designed to result in 50 percent and 80-90 percent maximum oxygen consumption were chosen for use during the hemodynamic study. Maximal oxygen consumption for the group estimated by this method was 18 ml O\textsubscript{2}/kg/min. TNG-O was titrated to a dose which lowered standing systolic blood pressure by at least 10 mm Hg (but not less than 95 mm Hg) and was associated with an increase in heart rate but did not produce orthostatic symptoms. The last dose given in the pretest period was at least the day prior to the hemodynamic study. Patients with aortic or mitral stenosis or chronic hypertension were excluded from the investigation. The study was reviewed and approved by the Clinical Investigation Committee of the Hershey Medical Center and all patients gave their informed consent for the use of nitroglycerin ointment and for the performance of the invasive procedures.

Hemodynamic studies were performed in the late morning after the patient had a clear liquid breakfast. An indwelling 20-gauge longdwell cannula was placed in the radial artery percutaneously. A No. 7 Fr Swan-Ganz thermodilution catheter was positioned in the pulmonary artery via venotomy from an antecubital vein. The position of the pulmonary catheter was manipulated until a pulmonary capillary wedge (PCW) pressure in the right lower lobe could be reproducibly obtained with inflation of the balloon at the tip of the catheter. Mean ($\text{MAP}$) and phasic systemic arterial, pulmonary arterial (PA), PCW, and right atrial (RA) pressures were recorded on an Electronics for Medicine VR 12 recorder with a CLC-1 computer and a rapid developer using Statham P23ID pressure transducers located at the midstch level when supine and 5 cm below the angle of Louis when upright. Heart rate was measured by electrocardiographic monitoring. Cardiac output was obtained by the thermodilution method using an Edwards Laboratory 9500 cardiac output computer and also recorded so that outputs could be verified independently. An average of at least three injections was made to obtain each cardiac output. Arterial and pulmonary artery blood samples were obtained simultaneously after cardiac output was measured and analyzed on a Lexington CO-Oximeter for determination of $O_2$ saturation and hemoglobin and calculations of arteriovenous oxygen differences ($A-V O_2$ ml O\textsubscript{2}/100 ml). Cardiac index and stroke volume index were calculated. The following formulae were utilized to make calculations: 1) systemic vascular resistance in dynes sec cm\textsuperscript{-5}, $SVR = \text{MAP} - \text{RA}) \times 80 \div \text{cardiac output}$; and 2) oxygen consumption in ml/min/m\textsuperscript{2}, $\text{VO}_2 = (\text{cardiac index}) \times A - \text{VO}_2 \times 10$. Arterial plasma catecholamines (norepinephrine and epinephrine) were determined by the radioisotopic method of Passon and Peuler.\textsuperscript{16} Arterial blood lactates were measured by the method of Hohorst.\textsuperscript{17} Catecholamine (2 ml) and lactate (1 ml) blood samples were obtained while the patients were lying supine at rest 20 minutes after placement of all catheters and at other times indicated in the protocol below. Blood samples for catecholamine determinations were placed in special tubes containing 30 lal of solution containing 5 mg of reduced glutathione and 9 mg of EDTA, pH 7.4. The tubes were kept in ice and were subsequently centrifuged under refrigeration for separation of cells and plasma. The plasma was then frozen until assayed shortly thereafter. Lactate specimens were placed in test tubes containing 0.1 ml of 70 percent perchloric acid and centrifuged under refrigeration. The supernatant was neutralized with potassium hydroxide and frozen until analyzed. Resting upright hemodynamics and blood samples were obtained after sitting comfortably on the bicycle ergometer for ten minutes.

Bicycle exercise was performed on an electronically-braked bicycle ergometer (Godart) at two successively higher work levels without interruption between workloads. Moderate and intense workloads were chosen so as to be approximately 50 and 80-90 percent of estimated maximum VO\textsubscript{2} (established at the time of pretesting). Pedal frequency was kept at 50-55 cycles/min by the patient by observation of an easily visible meter. In order to evaluate circulatory parameters in a steady state at each work level, pressure measurements, cardiac output determinations, and blood samples were obtained in a standard order after three minutes of cycling. The next work level began at the conclusion of these measurements. For each individual, the time for data collection before initiating the next level of exercise was kept as

CHEST, 76: 6, DECEMBER, 1979

UPTIGHT EXERCISE IN CONGESTIVE HEART FAILURE 641

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Table 1— Hemodynamic and Metabolic Data Before and After Nitroglycerin

<table>
<thead>
<tr>
<th>Stage</th>
<th>MAP (mm Hg)</th>
<th>HR (min⁻¹)</th>
<th>PCW (m/min/M²)</th>
<th>SVI (ml/min/M¹)</th>
<th>SVR (dyne sec/cm²)</th>
<th>A-V O₂ (ml O₂/100 ml)</th>
<th>VO₂ (mM/L)</th>
<th>Lactate</th>
<th>NE</th>
<th>EPI (pg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>102 ± 6.1</td>
<td>76 ± 4.8</td>
<td>19.4 ± 3.3</td>
<td>3.23 ± 38</td>
<td>44.7 ± 7.0</td>
<td>143 ± 233</td>
<td>4.99 ± .78</td>
<td>155 ± 23</td>
<td>0.67 ± .06</td>
<td>660 ± 236</td>
</tr>
<tr>
<td>Upright C</td>
<td>99 ± 6.9</td>
<td>81 ± 4.0</td>
<td>11.0 ± 2.5</td>
<td>2.45 ± 20</td>
<td>31.1 ± 3.7</td>
<td>1830 ± 211</td>
<td>6.67 ± .39</td>
<td>162 ± 12</td>
<td>0.82 ± .14</td>
<td>700 ± 217</td>
</tr>
<tr>
<td>TNG-O</td>
<td>91.5 ± 4.8</td>
<td>95.4 ± 8.4</td>
<td>9.1 ± 2.5</td>
<td>2.49 ± 21</td>
<td>26.7 ± 2.7</td>
<td>1703 ± 195</td>
<td>7.47 ± .50</td>
<td>182 ± 12</td>
<td>0.90 ± .10</td>
<td>791 ± 213</td>
</tr>
<tr>
<td>Moderate Ex C</td>
<td>118 ± 7.3</td>
<td>120 ± 6.8</td>
<td>18.0 ± 4.0</td>
<td>4.64 ± 47</td>
<td>39.4 ± 4.8</td>
<td>1089 ± 131</td>
<td>54 ± 1.17</td>
<td>2.35 ± .18</td>
<td>665 ± 95</td>
<td>129 ± 37</td>
</tr>
<tr>
<td>TNG-O</td>
<td>104 ± 6.0</td>
<td>125 ± 7.9</td>
<td>18.0 ± 3.4</td>
<td>4.89 ± 47</td>
<td>40.1 ± 4.7</td>
<td>906 ± 96</td>
<td>11.79 ± .77</td>
<td>538 ± 40</td>
<td>2.16 ± .37</td>
<td>717 ± 95</td>
</tr>
<tr>
<td>Intense Ex C</td>
<td>121.4 ± 8.5</td>
<td>140 ± 4.3</td>
<td>23.0 ± 3.3</td>
<td>5.07 ± 56</td>
<td>34.0 ± 3.9</td>
<td>1141 ± 229</td>
<td>13.58 ± .92</td>
<td>665 ± 82</td>
<td>4.46 ± .83</td>
<td>1915 ± 604</td>
</tr>
<tr>
<td>TNG-O</td>
<td>111 ± 7.5</td>
<td>152 ± 8.3</td>
<td>20.1 ± 3.4</td>
<td>5.50 ± 56</td>
<td>37.0 ± 4.4</td>
<td>942 ± 143</td>
<td>13.06 ± .82</td>
<td>714 ± 93</td>
<td>4.32 ± .56</td>
<td>1585 ± 426</td>
</tr>
</tbody>
</table>

Ex=exercise; C=control; TNG-O=nitroglycerin ointment; MAP=mean arterial pressure; HR=heart rate; PCW=mean pulmonary capillary wedge pressure; CI=cardiac index; SVR=systemic vascular resistance; A-V O₂=arteriovenous oxygen difference; VO₂=oxygen consumption; NE=norepinephrine; EPI=epinephrine. Values represent mean data ± standard error of the mean. One subject did not perform moderate exercise.

The following comparisons were made: upright control vs supine, upright TNG-O vs control, moderate exercise TNG-O vs control, intense exercise TNG-O vs control. A significant difference is indicated by a symbol beside the second parameter of the pair being compared (*=P<.005; †=P<.01; ‡=P<.001; §=P<.025).

RESULTS

There was no complication attributable to the application of TNG-O other than transient orthostatic hypotension in one patient which occurred at 60 minutes. By 90 minutes after TNG-O that patient could tolerate the upright position and bicycle exercise was performed without incident. Mean exercise time during control exercise for all patients averaged 6.8 minutes at the moderate level, and 6.6 minutes at the intense level. After TNG-O, mean exercise time at the moderate work load was 6.9 minutes, and 6.8 minutes at the intense level (P = NS versus control exercise times). No individual noted improvement in fatigue or dyspnea on exertion after use of nitroglycerin ointment. Table 1 contains the hemodynamic and metabolic data from each stage of the study.

Blood Pressure and Heart Rate

Mean arterial pressure tended to be lower at each stage of the study post nitroglycerin ointment (Fig 1). This tendency toward a lower mean blood pressure was statistically significant (P < .01) at the moderate exercise level. Diastolic blood pressure did not change significantly from the supine to upright position. Heart rate (Fig 1) tended to be greater during each part of the procedure after nitroglycerin ointment, reaching statistical significance only in the resting upright position (P < .005).

![Graph of Blood Pressure and Heart Rate](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21084/ on 04/28/2017)
Cardiac Index and Stroke Volume Index

Cardiac index averaged $3.23 \pm 0.38 \text{ L/min/M}^2$ supine and decreased to $2.45 \pm 0.20$ while sitting on the bicycle. After nitroglycerin ointment, cardiac index was significantly higher during both moderate ($4.64 \pm 0.47$ vs $4.89 \pm 0.47$ post TNG-O, $P < .025$) and intense ($5.07 \pm 0.56$ vs $5.50 \pm 0.50$, $P < .025$) exercise levels. At rest upright, cardiac index did not differ significantly pre and post TNG-O (Fig 2). Stroke volume index fell significantly ($P < .025$) with upright posture, but did not differ significantly before or after nitroglycerin at any point in the investigation subsequently.

Pulmonary Capillary Wedge Pressure

For the group, resting supine mean pulmonary capillary wedge pressure was elevated at 19.4 mm Hg. There was a significant drop in PCW pressure with the assumption of the upright posture (19.4 to 11.4 mm Hg, $P < .005$). PCW pressure tended to be slightly lower after use of nitroglycerin ointment at all times during the study (Fig 2). However, this finding did not reach statistical significance ($P > .05$) at any point.

Arteriovenous Oxygen Difference, Oxygen Consumption, and (A-V $O_2$)/CI

The arteriovenous oxygen difference widened significantly from supine to upright states ($P < .025$). After TNG-O, there was a further increase in the resting upright A-V $O_2$ ($6.76 \pm 0.39$ vs $7.47 \pm 0.50$, $P < .05$). At both levels of exercise (A-V $O_2$) tended to be lower post TNG-O, but this was not statistically significant. After TNG-O mean $V_O_2$ was greater in the upright position was essentially the same with moderate exercise, and was greater with intense exercise. However, the increased $V_O_2$ values were not of statistical significance. The ratio (A-V $O_2$)/CI was $2.94 \pm 0.39$ in the upright control period before TNG-O and $3.22 \pm 0.47$ after TNG-O ($P = \text{ns}$). With control exercise of moderate intensity, this ratio was $3.01 \pm 0.62$; after TNG-O it was $2.70 \pm 0.48$ ($P = \text{NS}$).
At the intense exercise level (A-V $O_2$)/CI decreased from $3.04 \pm .44$ in the control period to $2.57 \pm .29$ after TNG-O ($P < .05$).

**Metabolic Data**

Blood lactate levels rose progressively with exercise and peaked at the five-minute recovery period ($5.13 \pm .9$ mM/L pre TNG-O and $5.15 \pm .8$ mM/L post TNG-P, $P = .ns$) (Fig 3). Values in the resting position on the bicycle after TNG-O ($0.80$ mM/L) were slightly lower than the previously comparable measurements ($P > .05$). At no time during the study did blood lactate differ significantly pre or post TNG-O. Also, arterial pH did not differ significantly at any point before or after TNG-O.

Mean plasma norepinephrine was elevated at rest ($680$ pg/ml) supine and did not change significantly with the assumption of the upright position on the bicycle ($700$ pg/ml). Similarly, supine mean plasma epinephrine levels were elevated and did not change significantly ($P > .05$) while sitting on the bicycle. Both norepinephrine and epinephrine rose with exercise and peaked at the intense exercise workload. There were no statistically significant differences in either norepinephrine or epinephrine or total catecholamines pre or post TNG-O (Fig 3).

**DISCUSSION**

As a group, the patients in this study demonstrated an elevated supine left ventricular filling pressure with normal resting cardiac output. These findings reflect the clinical criteria of selecting patients with moderate heart failure for study and are generally in accord with the natural history of cardiac lesions causing impaired myocardial contractility and, eventually, an abnormal low cardiac index and symptoms at rest. Similarly, as a group, resting supine norepinephrine levels (when corrected for age) were elevated and are indicative of increased sympathetic tone compensating for impaired myocardial function. The lack of a statistically significant change in plasma catecholamines, heart rate, and diastolic blood pressure after assumption of the sitting position is consistent with increased tolerance to postural changes seen in heart failure. With one exception, it is generally considered that increases in PCW pressure with bicycle exercise (Fig 2) may be accepted as a reflection of heart failure. Although supine exercise may be more sensitive in disclosing potential left ventricular failure, upright exercise provides more information about the patient’s tolerance to daily life activities.

Information concerning the effects of long-acting or sustained-release nitrates upon upright exercise hemodynamics in patients with congestive heart failure is scant. Franciosa et al. found that a single $40$ mg dose of isosorbide dinitrate did not improve maximal exercise capacity or hemodynamics in patients with class 2-3 heart failure. Similarly, combination therapy of isosorbide dinitrate and hydralazine did not acutely improve exercise tolerance or maximal oxygen consumption in congestive heart failure patients. Borer et al., however, showed that sublingual nitroglycerin given to patients with rheumatic valvular disease increased maximal oxygen consumption and lowered pulmonary artery pressures in response to treadmill exercise. The aim of our investigation was to determine whether upright bicycle exercise could be safely performed and whether hemodynamics would be significantly improved with a longer acting nitroglycerin preparation. Further, we wished to determine whether there was any evidence of augmented nutritional blood flow to exercising muscles, such as increased systemic oxygen consumption, decreased peak lactate levels or decreased plasma catecholamine levels with exercise.

Compared to control exercise, pulmonary capillary wedge pressure tended to be lower after TNG-O and cardiac index (CI) were slightly higher; however, only the changes in CI were significant (Fig 2). CI increased in our patients an average of $0.26$ L/min/M$^2$ (5.3 percent increase) with moderate exercise and $0.43$ L/min/M$^2$ (8.5 percent increase) during intense exercise after TNG-O. Although both HR and SVI increased slightly with both levels of exercise after TNG-O, none of the changes was significant (moderate exercise: HR + 4.2, SVI + 1.8 percent; intense exercise: HR + 8.6 percent, SVI + 8.8 percent). Systemic $V_o_2$ was not significantly changed with TNG-O during exercise nor was A-V $O_2$ difference; however, the latter tended to widen less. The ratio (A-V $O_2$)/CI at intense exercise decreased significantly by 15.5 percent, suggesting that there was an increased circulatory reserve of the oxygen transport system after use of nitroglycerin ointment. Despite the significant changes noted in CI and (A-V $O_2$)/CI, the importance of these changes must be questioned since symptoms of exertional dyspnea and fatigue did not improve. Correlating with this lack of symptomatic improvement was the absence of changes in lactate production (an index of anaerobic metabolism). It has been suggested that the failure of the cardiovascular system to deliver adequate oxygenated blood to exercising skeletal muscles stimulates somatic afferent nerves and is at least one of the mechanisms by which the sympathetic nervous system responds excessively when heart failure patients exercise.

Total plasma catecholamines were greatly increased...
at the intense workload in our patients and this was not reduced by TNG-O. This gives further credence to our hypothesis that nitroglycerin does not significantly improve oxygen delivery to exercising skeletal muscle in heart failure. There is a preliminary report to suggest that in a volume overload rat model of congestive heart failure (aorto-caval fistula), intravenous nitroglycerin actually aggravated skeletal muscle blood flow during exercise. However, nitroglycerin did significantly attenuate the marked vasoconstriction in gut, renal, and cutaneous circulations.

Whether or not a similar situation occurs in humans is unknown. If it does, then TNG-O may protect visceral function when heart failure patients exercise.

The excessive sympathoadrenal response to exercise noted in heart failure can be considered a double-edged sword which could be protective or harmful to the exercising muscle. A recent study evaluated the effects of low levels of intraraerial norepinephrine in an isolated canine gracilis muscle preparation stimulated to contract statistically submaximally at 2.5 percent of maximal force. Low dose norepinephrine was shown to increase oxygen extraction (presumably by redistributing flow from inactive to active fibers) while flow was unchanged. This resulted in an increased oxygen consumption. However, higher levels of norepinephrine infusion resulted in a decrease in flow, and despite a further increase in oxygen extraction, a decreased oxygen consumption during exercise resulted. In severe congestive heart failure, excessive catecholamine liberation during strenuous exercise might reduce blood flow to exercising muscle groups and make them more hypoxic. It was hoped that an arterial dilating agent would improve nutritional flow to exercising muscle and that it might break this vicious circle. It is evident that the increase in cardiac index in our patients after use of TNG-O did not result in less release of catecholamines or an increase in systemic oxygen consumption or a reduction in lactic acidemia (Fig 3). It appears that the decrease in systemic vascular resistance seen in our patients after use of nitroglycerin ointment, although it did not have an adverse effect on the exercise response, did not result in increased nutritional flow to working muscle groups. It is possible that more severely ill patients or other vasodilators may demonstrate such an effect. Of equal importance is the fact that TNG-O did not produce any adverse effects in these patients during exercise. Therefore, TNG-O should be considered a safe and effective agent for improving hemodynamics and symptoms in the resting heart failure patient and does not impair exercise performance.

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REFERENCES


CHEST, 76: 6, DECEMBER, 1979

UPRIGHT EXERCISE IN CONGESTIVE HEART FAILURE 645
29 Longhurst J, Zelis R: Cardiovascular responses to local hindlimb hypoxemia—Relation to the exercise reflex. Am J Physiol (in press)

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The 18th Annual Seminar, Imageology 1980, will be held February 2-8 at the Fontainebleau Hilton Hotel, Miami Beach, and the course, Decision-Tree Approach to Diagnostic Radiology will be held February 8-11 at the Dutch Inn, Lake Buena Vista, Florida, under sponsorship of the Department of Radiology, Mount Sinai Medical Center, Miami Beach. For information, contact Ms. Lucy R. Kelley, c/o Miami Seminars, PO Box 943702, Coral Gables, Florida 33134.

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