Changes in bronchial and pulmonary arterial blood flow with progressive tension pneumothorax

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Carvalho, Paula, Jacob Hildebrandt, and Nirmal B. Charan. Changes in bronchial and pulmonary arterial blood flow with progressive tension pneumothorax, J. Appl. Physiol. 81(4): 1664–1669, 1996.—We studied the effects of unilateral tension pneumothorax and its release on bronchial and pulmonary arterial blood flow and gas exchange in 10 adult anesthetized and mechanically ventilated sheep with chronically implanted ultrasonic flow probes. Right pleural pressure (Ppl) was increased in two steps from −5 to 10 and 25 cmH2O and then decreased to 10 and −5 cmH2O. Each level of Ppl was maintained for 5 min. Bronchial blood flow, right and left pulmonary arterial flows, cardiac output (Q̇t), hemodynamic measurements, and arterial blood gases were obtained at the end of each period. Pneumothorax resulted in a 66% decrease in Q̇t, bronchial blood flow decreased by 84%, right pulmonary arterial flow decreased by 80% at Ppl of 25 cmH2O (P < 0.001). At peak Ppl, the majority of Q̇t was due to blood flow through the left pulmonary artery. With resolution of pneumothorax, hemodynamic parameters normalized, although abnormalities in gas exchange persisted for 60–90 min after recovery and were associated with a decrease in total respiratory compliance.

THE CARDIORESPIRATORY effects of tension pneumothorax have been well established in anesthetized and conscious animals (2, 8, 10, 12, 13). In spontaneously breathing goats and immature monkeys, unilateral tension pneumothorax results in elevated right-sided vascular and cardiac chamber pressures, systemic hypotension, and profound arterial hypoxemia (12). This apparent deterioration in cardiopulmonary function has previously been attributed to an impairment in venous return by positive pleural pressure (Ppl), with cardiovascular collapse indicated by decreases in cardiac output (Q̇r) and systemic arterial pressure (Psa). However, subsequent animal experiments by Rutherford et al. (12) and, more recently, by Gustman et al. (8) determined that cardiovascular collapse in response to tension pneumothorax is preceded by and likely caused by respiratory failure resulting in profound hypoxemia, hypercarbia, and subsequent acidemia. Although detailed studies of the cardiopulmonary phenomena associated with pneumothorax have provided considerable data, the alterations in bronchial arterial blood flow (Q̇br) and differential right and left pulmonary arterial flows (Q̇rpa and Q̇lpa, respectively) that occur with changes in Ppl and their contribution to the development of abnormalities in gas exchange have not been described. In addition, the physiological alterations that result when tension pneumothorax is released have not been systematically studied. The purpose of this study was, therefore, to determine the effects of progressive unilateral pneumothorax and its release on Q̇br on pulmonary arterial flow (Q̇pa) to each lung and on overall gas exchange.

Q̇pa has been shown to decrease with increased positive end-expiratory pressure (1). Because an increase in Ppl results in an elevation in total intrathoracic pressure, we hypothesized that progressive unilateral pneumothorax produces progressive decreases in both bronchovascular and ipsilateral pulmonary intravascular conductances. These, coupled with reduced Q̇t and driving pressures, would be accompanied by progressive decreases in the corresponding Q̇br and Q̇pa values. We also reasoned that progressive pneumothorax results in abnormalities in gas exchange that may be related to alterations in lung distensibility and surface forces.

A sheep model was chosen for this study because, as in the human, this species has an intact mediastinal pleura that allows production of unilateral pneumothorax. Furthermore, the pulmonary and bronchial circulatory systems of the sheep and their distribution to the pleura have been extensively studied and have been found to resemble those in the human (11).

METHODS

Surgical preparation. Ten adult sheep of mixed breeds (body weight 60–70 kg) were fasted for 12 h and then sedated with xylazine (0.5 mg/kg im). After induction of anesthesia with thiamylal sodium (15–20 mg/kg iv), the animals were intubated and anesthesia was maintained with 1–2% halothane. Supplemental O2 was provided to maintain arterial PO2 (Pao2) at >80 Torr, and the ventilator settings were adjusted to maintain arterial PCO2 (Paco2) at ~40 Torr. The rumen was vented via an orogastric tube.

Three ultrasonic flow probes (Transonic Systems) were implanted with sterile technique via a left thoracotomy through the fifth intercostal space. The bronchoesophageal trunk was identified, and the common bronchial branch was carefully dissected. One flow probe (2 mm) was placed around the common bronchial branch of the bronchoesophageal trunk for determination of Q̇br. Caution was taken to avoid compression or torsion of the vessel. The pericardium was then opened, and a second flow probe (16 mm) was placed around the pulmonary trunk for determination of Q̇t. To measure differential Q̇pa to each lung, the left hilum was dissected and a third flow probe (12 mm) was placed around the left pulmonary artery. For this, an extrapericardial approach was used to maintain pericardial tissue between the pulmonary trunk and left pulmonary arterial flow probes, thus preventing electrical interference between the two probes. The three flow probes were then tested by connecting them to an ultrasonic blood flowmeter (model T201, Transonic Systems).
to ensure that a satisfactory pulsatile reading was obtained. The three probe wires were passed through the chest wall and tunneled subcutaneously to exit at a point between the scapulae. A pleural tube (28-Fr, Argyle) was inserted to remove air from the pleural space, the ribs were approximated, and the skin was sutured in three layers. The lung was reexpanded by applying suction via the pleural tube, which was subsequently removed, and the animals were allowed to recover. Flow probe readings were obtained on a daily basis until they remained stable (7–10 days after implantation). At the end of this period, the flow probes placed around the pulmonary trunk produced consistent signals in all 10 animals, the bronchial arterial flow probes functioned in 9 of the 10 animals, and the left pulmonary arterial flow probes functioned consistently in 7 sheep. Accordingly, data on Qbr and vascular conductance are presented for 9 animals; data on differential Qpa values and conductances are presented for 7 animals; and data on arterial blood gases, airway pressure (Paw), Qt, and other hemodynamic parameters are presented for 10 animals.

On the day of the study, the animals were anesthetized, intubated, and ventilated as previously described. Anesthesia was maintained with 1–2% halothane. Supplemental O2 (inspired O2 fraction of 0.7) was given to maintain PaO2 at well above 100 Torr to minimize hypoxic pulmonary vasoconstriction and hypoxemia-induced changes in Qbr. The ventilator settings were adjusted to maintain PaCO2 at 45 Torr at the start of the experiment and were not subsequently altered. The rumen was vented via an orogastric tube as before. A Silastic catheter for arterial blood gas sampling and Psa measurement was placed in the left carotid artery. A balloon-tipped pulmonary artery catheter (8-Fr, Pentalumen Thermol dilution, Abbott) was placed via the left jugular vein and advanced into the pulmonary artery. A pleural tube (28-Fr, Argyle) was inserted into the right pleural space via an incision at the fifth intercostal space in the posterior axillary line. The tube tip was positioned in the most dependent part of the pleural space, secured with sutures, and tunneled subcutaneously to exit adjacent to the dorsal spine. Adjacent to the chest tube, a Silastic catheter (2.6 mm ID, 4.9 mm OD) was inserted in the pleural space and attached to a calibrated fluid-filled manometer for determination of Ppl (referenced to atmosphere). In three sheep, catheters were also inserted in a corresponding position in the left pleural space for determination of contralateral Ppl. An 18-gauge needle was inserted into the endotracheal tube and connected to a calibrated pressure transducer to monitor Paw. Psa and pulmonary arterial (Ppa) and pulmonary capillary wedge pressures (Pcw) were measured via calibrated transducers (referenced to the left atrium) and continuously recorded (model 2107–8890–00, Gould). A continuous chart recording of vascular flows as well as hemodynamic pressure parameters were obtained. Arterial blood samples were drawn under anaerobic conditions from the carotid catheter by using a heparinized syringe and were analyzed for pH, PaO2, and PaCO2 (model ABL-520, Radiometer).

Experimental protocol. With the anesthetized animals in the prone position, baseline arterial blood gas and physiological parameters were obtained. These included Qt, Qbr, Qlpa, Qrpa, mean Psa, end-inspiratory Paw, mean Ppa, and end-expiratory Pcw. Bronchovascular conductance (GbV; in ml·min⁻¹·Torr⁻¹) was calculated with

\[
G_{bV} = \frac{Q_{br}}{P_{sa} - P_{pa}}
\]

and total pulmonary vascular conductance (Gt; in l·min⁻¹·Torr⁻¹) was calculated with

\[
G_t = \frac{Q_T}{(P_{pa} - P_{cw})}
\]

We used Ppa as the downstream pressure for the bronchial circulation (4) and Pcw as the downstream pressure for the pulmonary bed. Conductance, the reciprocal of resistance, was calculated because the conductances for parallel vascular beds (such as the left and right lungs) are additive. Vascular conductance in each of the left and right pulmonary arteries was calculated by replacing Qt in the above equation by the respective Qpa values (in l/min).

End-expiratory Ppl on the right was increased in two steps, from −5 to 10 cmH2O and then to 25 cmH2O by introducing air via the pleural tube with a 1-liter syringe. Ppl was then returned in the same two steps by removing air from the pleural space. All experimental parameters stabilized at ~3–4 min after reaching each Ppl; therefore, arterial blood gases as well as physiological parameters were obtained at the end of each 5-min experimental period. After the experiment, Ppl was maintained at −5 cmH2O in three sheep to determine the length of time for stabilization to occur after resolution of unilateral tension pneumothorax.

Statistical analysis. The changes in arterial blood gases and physiological parameters were compared with the respective baseline values in each group by one-way analysis of variance followed by Dunnett's test. P < 0.05 was considered significant. All data are presented as means ± SE.

RESULTS

Effects on Qt, differential Qpa values (Fig. 1), and conductances (Table 1). Qt progressively decreased from a mean baseline value of 3.5 ± 0.35 l/min to approximately one-third of baseline values as Ppl was raised by 10.2 ± 0.33 l/min from −5 to 25 cmH2O (n = 10; P < 0.001) but then returned to baseline as Ppl normalized. With a right-sided pneumothorax, Qrpa at Ppl of 25 cmH2O decreased to one-fifth of its baseline value (n = 7; P < 0.001) and then returned to baseline flow with normalization of Ppl. When related to Qt, Qrpa decreased from 57 ± 6% at baseline to 28 ± 6% at Ppl of 25 cmH2O (P <
Effects of pleural pressure on pulmonary arterial conductances

<table>
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<tr>
<th>Ppl, cmH₂O</th>
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<td>25</td>
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Values are means ± SE; n = 7 sheep. Ppl, pleural pressure; Gipa, left pulmonary arterial conductance; Grpa, right pulmonary arterial conductance. *Significantly different compared with Gipa values, P < 0.05.

0.001), then approximated baseline values when Ppl normalized. Gipa did not change significantly with increasing Ppl, although Gipa/Qr significantly increased from a baseline value of 43 ± 6 to 72 ± 6% at Ppl of 25 cmH₂O (n = 7; P < 0.001), then returned to 50 ± 5% of total flow with resolution of pneumothorax.

GT decreased when Ppl increased from −5 to 25 cmH₂O (n = 7; P < 0.05), then approximated baseline values when pneumothorax was released. Left pulmonary arterial conductance did not significantly change at Ppl of 25 cmH₂O compared with baseline values, whereas right pulmonary arterial conductance decreased to one-third of baseline values at peak Ppl (n = 7; P < 0.05). Both conductances returned to baseline at Ppl of −5 cmH₂O (Table 1).

Effects on Qbr and vascular conductance. Qbr decreased from 46.5 ± 17 ml/min at baseline to approximately one-sixth of baseline values at Ppl of 25 cmH₂O (Fig. 2A; n = 9; P < 0.001). With resolution of pneumothorax, Qbr rose to 145 ± 15% of baseline values (P < 0.05). Gbv varied greatly among animals, predominantly because of large variations in Qbr. The overall trend, however, showed an initial increase in Gbv after Ppl increased to 10 cmH₂O, followed by a decrease to ~45% of baseline values at 25 cmH₂O (Fig. 2B; n = 9).

Effects on arterial blood gases. Figure 4 shows the relationship between PAcO₂ and pH during the development of tension pneumothorax and during recovery. PAcO₂ increased from 42 ± 2 Torr at baseline to 50 ± 5 Torr at Ppl of 25 cmH₂O (n = 10; P < 0.001) and then continued to increase as Ppl returned to normal, ending
with a PaCO₂ of 55 ± 6 Torr at Ppl - 5 cmH₂O (P < 0.001). Accordingly, arterial pH decreased from a mean value of 7.46 ± 0.02 at baseline to 7.38 ± 0.04 at Ppl of 25 cmH₂O, then remained at 7.37 ± 0.03 when Ppl decreased to -5 cmH₂O (P < 0.015). With the increase in Ppl, the animals developed worsening acidemia that did not resolve acutely when Ppl returned to baseline but approximated baseline values 60–90 min after resolution of pneumothorax. PaO₂ decreased from 150 ± 28 Torr at baseline to 59 ± 12 Torr at Ppl of 25 cmH₂O (n = 10; P < 0.001), then decreased further to 55 ± 9 at Ppl of 10 cmH₂O during release of pneumothorax (P < 0.001), followed by a mild increase to 63 ± 9 Torr when Ppl returned to baseline (P < 0.05). After a stabilization period of 60–90 min in three animals, PaO₂ increased to 95 ± 13 Torr.

Effects on respiratory compliance (CL). Total CL decreased to approximately one-third of baseline values at peak Ppl (Fig. 5). CL did not return to baseline once Ppl normalized; instead it remained at ~60% of baseline (n = 6; P < 0.05).

Fig. 4. Relationship between arterial PCO₂ (PaCO₂; in Torr) and pH during development of tension pneumothorax and during recovery. Values are means ± SE. B₁, baseline data obtained at Ppl of -5 cmH₂O prepneumothorax (n = 10 animals); n = 10 for remainder of data points at Ppl of 10 cmH₂O up through -5 cmH₂O down. B₂, data obtained 60–90 min after resolution of pneumothorax (n = 3). In HCO₃⁻-pH diagram, uncompensated changes follow a line with a slope of 10 slykes (10sl).

Effects on contralateral Ppl values. In this model, the contralateral (left) Ppl at end-expiration (Ppl_c) in three sheep varied as follows, compared with Ppl in the experimental side. At Ppl of -5, 10, and 25 cmH₂O, Ppl_c = -5 ± 1, 3 ± 1, and 7 ± 2 cmH₂O, respectively. As the pneumothorax was released, Ppl_c followed the same trend, with Ppl_c = 4 ± 1 and -5 ± 2 cmH₂O at Ppl of 10 and -5 cmH₂O, respectively.

Time for resolution of hemodynamic and gas-exchange abnormalities. In three animals, hemodynamic parameters and arterial blood gases were serially obtained at Ppl of -5 cmH₂O after resolution of pneumothorax. In two of the three animals hemodynamic parameters, PaO₂, PaCO₂, and pH resolved within 60 min, whereas in the third animal these parameters had approximated baseline values at ~90 min. The data obtained from these three animals showed the same trend and were not significantly different from those of the remaining seven animals at all Ppl values studied.

DISCUSSION

The cardiovascular responses to tension pneumothorax have previously been well described by numerous investigators (2, 8, 10, 12, 13, 16). However, the effects of pneumothorax on Qbr, differential Qrpa and Qlpa values, and their respective conductances have not been previously described. In addition, the physiological effects that result when tension pneumothorax is progressively released had not been systematically studied. Therefore, in the present study, we investigated the effects of increasing then decreasing Ppl on Qbr, Qpa to each lung, and gas exchange in adult anesthetized sheep receiving positive-pressure ventilation. We chose an end-expiratory Ppl range of -5 to 25 cmH₂O because this range has previously been reported clinically in association with tension pneumothorax (9).
With the induction of tension pneumothorax in our model, QT decreased by nearly 70% when end-expiratory Ppl reached its peak of 25 cmH2O, then rapidly returned to baseline as the pneumothorax was released (Fig. 1). This was accompanied by a 40% decrease in Psa that quickly reversed when Ppl returned to baseline (Fig. 3). The decrease in QT demonstrated in our study is in accordance with that found by Culver et al. (6) in anesthetized mechanically ventilated dogs. In their study, increases in Ppl in a closed-chest preparation resulted in a decrease in QT without evidence of ventricular dysfunction. However, other investigators (2, 8, 12) have found that QT and Psa in spontaneously breathing animals did not decrease with the development of tension pneumothorax. Rutherford et al. (12) found that QT, as the product of a rising heart rate and falling stroke volume, was maintained at or above control levels after tension pneumothorax developed in spontaneously breathing goats and monkeys. In their study, QT continued to sustain Psa for a period of time after respirations ceased. These differences in results can be explained by the fact that the animals in our study were not spontaneously breathing and therefore could not generate a negative intrathoracic pressure at any time during the respiratory cycle. Indeed, Gustman et al. (8) found that mechanically ventilated animals had a decrease in QT and a trend for a decrease in Psa. By contrast, animals breathing spontaneously developed a negative Ppl during part of the respiratory cycle and were able to maintain QT and Psa.

Qbr decreased by >80% with increasing Ppl (Fig. 2A). It is possible that elevated left-sided filling pressures, as evidenced by a near-doubling in Pcw, may have contributed to the decrease in Qbr (Fig. 3). Our findings are, therefore, in agreement with those of Wagner et al. (14) and Charan et al. (4), who showed that increases in left atrial pressure cause increases in bronchial vascular resistance, suggesting that the bronchial vasculature has autoregulatory mechanisms that are possibly of a myogenic nature.

The determinants for Qbr are Psa (upstream pressure), mean Ppa (downstream pressure), and bronchovascular resistance (4). When Ppl increased from −5 to 10 cmH2O, Qbr initially decreased without a concomitant decrease in Gbv (Fig. 2, A and B). When Ppl further increased to 25 cmH2O, there was a marked decrease in Qbr associated with further decreases in both Psa and Ppa, thus resulting in a decrease in Gbv.

In our study, tension pneumothorax produced an increase in Paw that may, in turn, have influenced Gbv. However, Paw remained elevated after resolution of pneumothorax, possibly because of persistent alveolar collapse and bronchoconstriction resulting from mediators such as serotonin and histamine that are released as a result of lung distortion and collapse (15). It may also be speculated that a decrease in alveolar surfactant production resulted in persistently increased end-inspiratory Paw values. When Ppl returned to baseline, there was a trend for higher Qbr compared with prepneumothorax flows despite a concurrent trend toward higher Paw. This increase in Qbr over baseline may have been a function of changes in arterial blood gases. It has been shown that Qbr increases in response to hypoxemia and that this effect is independent of changes in QT and Psa (3). In our study, the animals developed a significant degree of arterial hypoxemia with induction of pneumothorax and remained acutely hypoxic despite its resolution. In addition, previous studies have shown that hypercarbia can cause an independent increase in Qbr (3). In our study, because the animals were anesthetized and mechanically ventilated with a fixed minute ventilation, they were unable to compensate for a decrease in effective alveolar ventilation secondary to the pneumothorax. As result, a significant degree of hypercarbia developed with progression of pneumothorax that did not acutely resolve with return of Ppl values to baseline. This persistent increase in PαCO2 may thus also have contributed to the increase in Qbr.

The abnormalities in acid-base homeostasis with tension pneumothorax are depicted in Fig. 4. With the initial increase in Ppl from −5 to 10 cmH2O, a predominantly metabolic acidosis ensued; with a further increase to 25 cmH2O, however, respiratory acidosis resulted. The slope of the curve between these two points is in the vicinity of −30°, which is suggestive of an acute respiratory acidosis where there has been insufficient time for the intravascular and interstitial compartments to equilibrate (7). When Ppl decreased to 10 cmH2O and, subsequently, to −5 cmH2O, PαCO2 continued to rise and the slope of the curve remained at approximately −30°, indicating a worsening respiratory acidosis. When equilibration occurred at Ppl of −5 cmH2O after 60–90 min, the slope of the curve paralleled the 10 slykes (sl) line, which represents recovery of equilibration of the intravascular and interstitial compartments. It is surprising that, after the increase in Ppl from 10 cmH2O, there appears to be a lack of metabolic contribution to the resultant acidosis. We do not have an explanation for this finding; in view of the decrease in QT and worsening systemic perfusion resulting from tension pneumothorax, a large metabolic component in addition to the evident respiratory acidosis would have been expected.

With an increase in Ppl, alveolar ventilation decreased and hypoxemia and hypercarbia worsened. These did not improve acutely as Ppl normalized, although there was slight improvement in gas exchange after a period of stabilization at Ppl of −5 cmH2O. Despite this period of stabilization, however, PαO2 and PαCO2 did not return to baseline values, indicating the persistence of ventilation-to-perfusion inequalities. These findings are in agreement with those of Bennett et al. (2), who studied progressive pneumothorax in dogs and concluded that the hypoxemia that occurs with pneumothorax appears to be the result of several factors such as hyperventilation, ventilation-to-perfusion nonuniformities, and intrapulmonary shunting. In our model, tension pneumothorax appears to have resulted in localized alveolar collapse and consequent localized alveolar hypoxia on the affected side resulting in shunting of blood and, hence, worsening oxygenation and ventilation. Cl decreased with increasing Ppl and remained significantly lower.
than baseline values when Ppl returned to $-5$ cmH$_2$O, despite normalization of Qpa, Psa, and Qt (Fig. 5). This persistent abnormality in Cl appears to correlate with the persistence in gas-exchange abnormalities.

With a right-sided pneumothorax, the majority of Qt was due to flow remaining in the left lung. Ppa increased as previously described (2, 5, 8, 12, 16). Conductance in the right pulmonary artery decreased to one-third of baseline values, whereas conductance in the left arterial system did not significantly change. There could be two potential explanations for this. First, because the stimulus-response curve of pulmonary vasomotor constriction is not linear and marked vasomotor constriction occurs when the alveolar oxygen tension is $<70$ Torr (15), it is possible that right-sided tension pneumothorax contributed to localized alveolar hypoxia, a consequent increase in Ppa, and a decrease in conductance in the right pulmonary arterial bed. A second explanation for this differential decrease in conductance is due to mediastinal shift and mechanical collapse of pulmonary vessels as a result of pneumothorax (2). In this model, the second mechanism appears to be the predominant one because, although Pao$_2$ did not return to baseline once Ppl normalized, the increase in mean Ppa resolved and vascular conductances returned to baseline. These findings therefore suggest that in this model of tension pneumothorax, vascular narrowing due to mechanical factors is the predominant mechanism of elevated pulmonary vascular pressures and decreased conductance in the right pulmonary arterial bed.

In the studies of Rutherford et al. (12) and Gustman et al. (8), the investigator attributed the cardiovascular collapse that results from tension pneumothorax to respiratory failure. In these two studies, pneumothorax was maintained, in contrast to ours, where it was released. In our study, hemodynamic parameters normalized with resolution of pneumothorax, although hypoxemia and hypercarbia persisted. In the study of Rutherford et al. (12), pneumothorax was released in only one animal; although blood gas data were not provided for this animal, hemodynamic parameters normalized with the return of Ppl to normal. It is possible that a more prolonged duration of gas-exchange impairment is necessary for cardiovascular collapse to occur and that hemodynamic collapse would have ensued if the pneumothorax in our study had not been released. It is also possible that a combination of gas-exchange impairment and pulmonary vascular narrowing due to mechanical factors is necessary for cardiovascular collapse to occur.

In summary, this study demonstrated that unilateral tension pneumothorax produced a decrease in Qbr disproportionate to the decrease in Qt; this decrease may have been secondary to an increase in Paw or, perhaps, to an acute elevation in left-sided filling pressures. When Ppl values returned to baseline, trends for higher Qbr and Gbv values compared with baseline were observed (despite a trend for a persistent elevation in Paw) that may have resulted from persistent hypoxemia and hypercarbia or from release of vasoactive mediators during vascular reperfusion. Tension pneumothorax produced a decrease in vascular conductance in the ipsilateral pulmonary artery. Although localized alveolar hypoxia may have contributed to these effects, pulmonary vascular narrowing due to mechanical factors appeared to be the predominant mechanism responsible for the decrease in conductance. Pneumothorax also resulted in ventilation-to-perfusion inequalities that persisted despite the return of Ppl to normal. These residual abnormalities in gas exchange after pneumothorax were associated with a decrease in total Cl. Thus we conclude that increases in intrathoracic pressure produced by unilateral tension pneumothorax result in progressive decreases in Qbr and ipsilateral Qpa accompanied by persistent abnormalities in gas exchange.

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