anesthesia took 15 minutes to go from T_{2-3} to C_{2-3} once it started to rise would be consistent with an epidural anesthetic containing a long-acting agent with slow onset, such as bupivacaine. The inability to aspirate cerebrospinal fluid via the epidural catheter at that time would also be consistent with a slow-onset epidural anesthetic.

We feel that the cause of delayed-onset total spinal anesthesia in this patient most likely to have been subarachnoid migration of the catheter tip following epidural injection of the anesthetic solution, thus allowing the anesthetic solution to "leak" slowly from the epidural space to the subarachnoid space. The inability to aspirate cerebrospinal fluid from the catheter while the patient was in the operating room may have been due to kinking of the catheter while the patient was in the supine position and lying on the catheter.

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Circulatory Changes during Direct Laryngoscopy and Tracheal Intubation:

Influence of Duration of Laryngoscopy with or without Prior Lidocaine

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Circulatory stimulation during tracheal intubation results from both direct laryngoscopy and placement of the tube in the trachea. Pharmacologic attempts to attenuate these blood pressure and heart rate (HR) elevations have included topical anesthesia of the oropharynx,[1,2] iv lidocaine,† adrenergic blocking drugs,[3,4] vasodilating drugs,[5] and deep anesthesia.[6] Although these approaches have been partially successful, we have observed that any protection provided against blood pressure increases may be negated by prolonged laryngoscopy. The influence of the duration of laryngoscopy on circulatory changes during laryngoscopy and tracheal intubation has not been examined. This report describes such changes during direct laryngoscopy lasting 60 seconds followed by tracheal intubation with or without laryngotracheal, intravenous or topical oropharyngeal administration of lidocaine.

Methods

Thirty-six adult patients without known heart disease and scheduled for major noncardiac operations were studied. The protocol was approved by the Indiana University School of Medicine Clinical Research Committee, and patient consent was obtained. Preanesthetic medication consisted of im administration of morphine (8–15 mg) and scopolamine (0.4 mg). Upon arrival of the patient in the operating room a radial-artery catheter was inserted to permit continuous recording of mean arterial pressure (MAP) and calculation of HR.

While the patient was breathing oxygen, *d*-tubocurarine (*d*Tc, 40 μg/kg) was administered iv, followed 3 minutes later by thiamylal (4 mg/kg) and succinylcholine (SCh, 2 mg/kg). Direct laryngoscopy with a straight laryngoscope blade was initiated 1 minute after thiamylal–succinylcholine and maintained for

Table 1. Comparative Patient Data from the Study Groups (Mean ± SE)

	Control	Viscous Lidocaine	Intravenous Lidocaine
Age (years)	47 ± 3	51 ± 2	49 ± 2
Weight (kg)	75 ± 3	84 ± 5	76 ± 3
Mean arterial pressure (torr)			
Awake	92 ± 2	92 ± 3	97 ± 3
1 minute after thiamylal–SCh	82 ± 3*	80 ± 4*	84 ± 4*
Heart rate (beats/min)			
Awake	72 ± 4	71 ± 4	75 ± 4
1 minute after thiamylal–SCh	83 ± 5*	85 ± 3*	83 ± 4

* $P < 0.05$ compared with awake value.

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† Denlinger JK, Messner JT, D'Orazio DJ, et al: Effect of intravenous lidocaine on the circulatory response to tracheal intubation. Abstracts of Scientific Presentations, Annual Meeting of the American Society of Anesthesiologists, October 1975, pages 43–44.

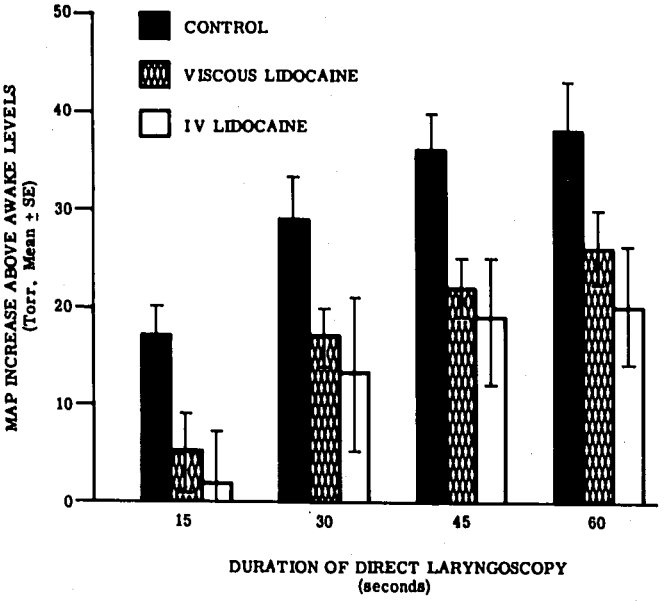

FIG. 1. Mean arterial pressure (MAP) increased progressively above awake levels after 15, 30, 45, and 60 seconds of direct laryngoscopy. MAP increases were significantly greater ($P < 0.05$) at all times in the control patients compared with the lidocaine-treated groups. The difference between blood pressure increases with viscous and intravenous lidocaine was not significant.

60 seconds. The tracheal tube was placed after 60 seconds of laryngoscopy. Arterial blood for blood-gas determinations was obtained at the beginning and end of laryngoscopy from all patients. Ventilation with oxygen was continued for 1 minute after tracheal intubation.

The patients were divided into three treatment groups, with 12 patients in each category. Control patients received only laryngotracheal administration of lidocaine (2 mg/kg) just before placement of the tracheal tube. In the second group, viscous lidocaine (25 ml, 2 per cent) was utilized as a mouthwash and gargle 10 minutes before anesthetic induction. Any residual viscous lidocaine was swallowed at the conclusion of the gargle. Laryngotracheal administration of lidocaine was carried out as in the control patients. Lidocaine, 1.5 mg/kg, was administered iv to the remaining 12 patients 90 seconds before the start of laryngoscopy. These patients did not receive laryngotracheal administration of lidocaine.

Data were analyzed with Student's t test, and $P < 0.05$ considered significant.

RESULTS

Patient ages, weights, and awake MAP and HR values were not different among the three treatment groups (table 1). MAP's and HR's 1 minute after thiamylal–SCh and just before starting laryngoscopy were also similar (table 1). Pa_{O_2} decreased from 356 ± 23 torr (mean ± SE) at the start of laryngoscopy to 296 ± 44 torr 60 seconds later. During the same period Pa_{CO_2} increased from 38 ± 2 to 44 ± 3 torr.

Mean arterial pressure increased progressively with the duration of laryngoscopy, but the average increase above awake levels was always significantly greater ($P < 0.05$) at 15, 30, 45, and 60 seconds in the control than in the viscous or intravenous lidocaine-treated patients (fig. 1). The magnitudes of MAP elevations in the two lidocaine-treated groups were not significantly different. Near-maximal pressor responses were present in all three groups by 45 seconds. Compared with awake measurements, MAP was significantly increased ($P < 0.05$) after 15 seconds of laryngoscopy in the control patients and after 30 seconds in the two lidocaine-treated groups.

The protective effect of viscous or intravenous lidocaine treatment was further apparent when the numbers of patients in whom MAP increases to more than 20 torr above awake levels developed were considered (fig. 2). At each interval during laryngoscopy, more patients in the control group than in the viscous and intravenous lidocaine treatment groups had MAP increases of more than 20 torr.

Mean arterial pressure increased further after tracheal intubation, and the magnitude and time of maximal increase were related to the use of laryngotracheal administration of lidocaine (table 2). Tracheal intubation resulted in less than a 10-torr additional blood pressure increase in each of the two groups receiving laryngotracheal administration of lidocaine. This maximal pressure increase occurred within 10

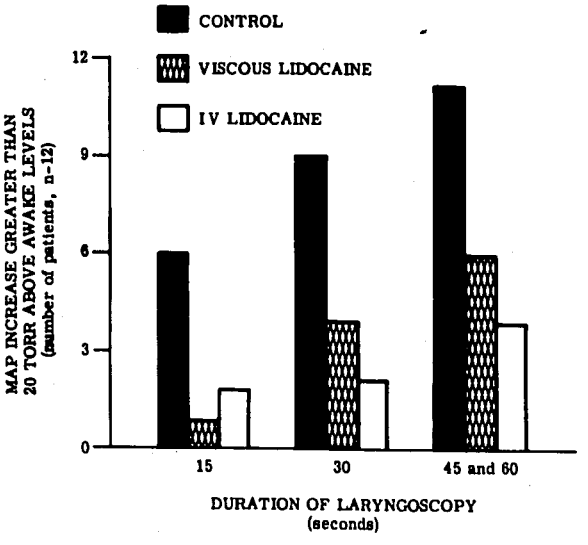

FIG. 2. More control than lidocaine-treated patients had MAP increases of more than 20 torr after 15, 30, 45, and 60 seconds of direct laryngoscopy.

seconds after placement of the tracheal tube, and MAP was decreasing spontaneously toward awake levels by 3 minutes after thiamylal–SCh. In contrast, MAP increased an additional 22 ± 2 torr ($P < 0.05$) when the tracheal tube was placed in patients receiving intravenous but not laryngotracheal administration of lidocaine. This maximum elevation in these patients occurred 25 ± 6 seconds after tracheal intubation and persisted 3 minutes after thiamylal–SCh.

Heart rates increased similarly in all groups after thiamylal–SCh, as well as in response to tracheal intubation (tables 1 and 2). The total HR increase during laryngoscopy and intubation (*i.e.*, 2 minutes after thiamylal–SCh) was 20–30 beats/min. This increase in HR was not significantly different between groups. However, HR decreased spontaneously 3 minutes after thiamylal–SCh only in the two groups receiving laryngotracheal administration of lidocaine.

TABLE 2. Circulatory Changes during Direct Laryngoscopy and Following Tracheal Intubation (Mean ± SE)

	Control and Laryngotracheal Lidocaine	Viscous Lidocaine and Laryngotracheal Lidocaine	Intravenous Lidocaine without Laryngotracheal Lidocaine
Mean arterial pressure			
After 60 seconds of direct laryngoscopy (torr)	130 ± 5	118 ± 4	114 ± 7
Maximal value after tracheal intubation (torr)	136 ± 5	123 ± 4	136 ± 7*
Time of maximal increase following tracheal intubation (seconds)	10 ± 3	7 ± 2	25 ± 6†
3 minutes after thiamylal–SCh (torr)	117 ± 5*	109 ± 3*	132 ± 5†
Heart rate (beats/min)			
2 minutes after thiamylal–SCh	99 ± 4	92 ± 3	95 ± 5
3 minutes after thiamylal–SCh	88 ± 4	87 ± 4	95 ± 3

* $P < 0.05$ compared with preceding MAP or HR value.
† $P < 0.05$ compared with control and laryngotracheal lidocaine group.

DISCUSSION

These data demonstrate progressive increases in MAP above awake levels during the first 45 seconds of laryngoscopy. Prolonging laryngoscopy to 60 seconds produced less than a 5-torr additional increase in MAP above the value after 45 seconds. The blood pressure response was attenuated but not prevented by either oropharyngeal topical or intravenous administration of lidocaine. The value of laryngotracheal administration of lidocaine just before tracheal intubation was demonstrated by the minimal additional MAP increases (10 torr or less) in response to tracheal intubation. When laryngotracheal administration of lidocaine was not utilized MAP increased an additional 22 torr with placement of the tracheal tube. In contrast, a previous report demonstrated that laryngotracheal administration of lidocaine did not attenuate the blood pressure response to laryngoscopy and intubation requiring about 30 seconds.[7] However, the present data demonstrate that the increase of MAP is not maximal until after 30 to 45 seconds of laryngoscopy. Therefore, it may not be possible to separate the relative contributions of laryngoscopy and tracheal intubation to the total pressor response when laryngoscopy takes less than 30 to 45 seconds. When laryngoscopy is prolonged beyond 45 seconds, the MAP increase is nearly complete and the contribution of subsequent tracheal intubation and influence of laryngotracheal administration of lidocaine become apparent.

Heart rate also increased during laryngoscopy, but the magnitudes of the increases were not significantly different among the three treatment groups studied. However, the value of laryngotracheal administration of lidocaine is again suggested by the spontaneous reductions in HR towards awake levels only in the groups receiving this treatment. Similarly, King *et al.*[5] found that deep anesthesia attenuated the blood pressure, but not HR, response to intubation. The reason for this separation of blood pressure and HR responses during tracheal intubation is unknown.

The protective effect of intravenous administration of lidocaine (1.5 mg/kg 90 seconds before beginning laryngoscopy), reported earlier* and confirmed by this study, may reflect additional central nervous system depression (*i.e.*, deeper anesthesia) produced by the local anesthetic.[8] In contrast, topical oropharyngeal anesthesia with viscous lidocaine would seem more specific, as this should anesthetize those areas in contact with the laryngoscope blade. Stimulation of these areas would seem the most likely explanation for blood pressure elevations during laryngoscopy. Serum lidocaine concentrations remain below therapeutic levels (2–5 μg/ml) following use of viscous lidocaine.[2]

Attempts to attenuate pressor responses to laryngoscopy and intubation would seem to be most appropriate when intubation is likely to take more than 30 seconds. This is particularly important in patients with coronary-artery disease or intracranial hypertension. Although viscous and intravenously administered lidocaine were equally protective, the more specific production of anesthesia in areas stimulated by direct laryngoscopy would seem to make prior oropharyngeal anesthesia the more logical choice. When laryngoscopy is prolonged, laryngotracheal

administration of lidocaine is necessary to attenuate the pressor response to intubation and speed the spontaneous decreases in both blood pressure and HR after placement of the tube in the trachea.

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Mepivacaine in Amniotic Fluid Following Maternal Epidural Anesthesia

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Amniotic fluid, with a volume at term between 500 and 2,000 ml[1], may be an important compartment for drug distribution between mother, placenta, and fetus. There is dynamic exchange of content such as water and electrolytes across the amnion and chorion with maternal serum.[2] Its composition is also affected by fetal micturition and swallowing. The fetus appears to void *in utero* every 50 to 155 minutes in amounts of as much as 30 ml per hour,[3] and swallows an equal amount.[4,5]

As part of our studies of perinatal pharmacology of local anesthetics, we have measured the concentration of mepivacaine in amniotic fluid following maternal epidural anesthesia for labor and delivery.

Materials and Methods

Thirteen healthy parturients at term were studied after informed consent was obtained. When labor had become active, amniotic membranes were ruptured artificially under antiseptic conditions. A plastic intrauterine catheter¶ was inserted approximately 6 inches within the uterus, using a transcervical approach. The loss of amniotic fluid did not appear great following amniotomy in any of these patients. The lumbar epidural catheter was introduced, usually prior to or shortly after the application of the intrauterine catheter. Anesthesia was established and maintained with mepivacaine (1.5 or 2.0 per cent without epinephrine) by intermittent injection through the indwelling epidural catheter.

Six to 10 ml of amniotic fluid were aspirated from the intrauterine catheter using a syringe at intervals ranging from 10 to 60 minutes. Collection of this fluid began as soon as the catheter was inserted. The initial 5 ml of amniotic fluid were discarded after the first sample, since this volume represented the dead space within the catheter lumen.

Table 1. Characteristics of the Study Population

Maternal age (years)	25 ± 1.5*
Maternal height (inches)	64 ± 0.6
Maternal weight (pounds)	145 ± 6.0
Total dose of local anesthetic (mg)	451 ± 57
Time from initial dose of local anesthetic to delivery time (minutes)	218 ± 31
Birth weights of babies (g)	3378 ± 139
Apgar scores	
1 min	9 (6–9)†
5 min	9 (8–10)
Volume of gastric content (ml)	5.5 ± 1.3

* Mean ± standard error.
† Median (range).

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