3. Kumazawa T: Wolff-Parkinson-White syndrome and anesthesia. Jpn J Anesthesiol 19:68–73, 1970
4. Gallagher JJ, Svenson RH, Sealy WC, et al: The Wolff-Parkinson-White syndrome and the preexcitation dysrhythmias. Med Clin North Am 60:101–123, 1976
5. Hannington-Kiff JG: The Wolff-Parkinson-White syndrome and general anaesthesia. Br J Anaesth 40:791–795, 1968
6. Narula OS: Symposium on Cardiac Arrhythmias (Part 4). Wolff-Parkinson-White syndrome. Circulation 47:872–887, 1973
7. Delineau MA, Meriel P: Repercussions de l'anesthesia generale sur le syndrome de Wolff-Parkinson-White. Cahiers d'Anesthesiologie 19:7–14, 1971
8. Iga E: Anesthetic management of the WPW syndrome. Jpn J Anesthesiol 23:357–364, 1973
9. Yusa T, Sugiu T, Sato T: Neuroleptanesthesia for a myasthenic patient with WPW syndrome. Jpn J Anesthesiol 21:498–504, 1972
10. Göthert M, Wendt J: Inhibition of adrenal medullary catecholamine secretion by enflurane. ANESTHESIOLOGY 46:400–410, 1977
11. Göthert M, Dreyer C: Inhibitory effect of halothane anaesthesia on catecholamine release from the adrenal medulla. Naunyn-Schmiedebergs Arch Pharmacol 277:253–266, 1973
12. Anderson EJ, Rosenthal MH: Pancuronium bromide and tachyarrhythmias. Crit Care Med 3:13–15, 1975

Anesthesiology
51:556–558, 1979

LDH_5 Changes after Cholecystectomy or Hysterectomy in Patients Receiving Halothane, Enflurane, or Fentanyl

OSCAR VIEGAS, M.D.,* AND ROBERT K. STOELTING, M.D.†

The LDH_5 isoenzyme fraction of lactate dehydrogenase is felt to be relatively specific for hepatocellular injury.[1] Klar *et al.*[2] measured LDH_5 changes in the first 24 hours after elective cholecystectomy and reported this isoenzyme increased more and remained elevated longer after halothane than after methoxyflurane or thiopental–meperidine anesthesia. They suggested their data were consistent with a selective hepatotoxic effect produced by halothane. In contrast, another report failed to document a detrimental effect of halothane when administered to patients undergoing cholecystectomy.[3]

In view of these conflicting reports, we elected to repeat the study of Klar *et al.* by again determining LDH_5 values before and after elective cholecystectomies performed with halothane–N_2O anesthesia. In addition, LDH_5 measurements were extended to include an additional procedure (hysterectomy) and two other commonly used anesthetic drugs (enflurane and fentanyl).

METHODS

Sixty nonobese adult patients undergoing elective cholecystectomy with intraoperative cholangiography (30 patients) or abdominal hysterectomy (30 patients) were studied. The study protocol was approved by the Indiana University School of Medicine Human Research Committee. No patient had a history of hepatic disease, was taking drugs known to alter hepatic function, or had previously received a halogenated anesthetic. Preanesthetic medication was with diazepam, 5–10 mg, orally, and atropine, 0.4 mg, im, 60 to 90 min prior to operation. Anesthetic induction was with *d*-tubocurarine, 40 μg/kg, followed by thiamylal, 4 mg/kg, and succinylcholine, 1.5 mg/kg, to facilitate tracheal intubation. Anesthesia was maintained with 60 per cent inspired nitrous oxide and 0.5–1 per cent halothane (20 patients), 0.5–1.5 per cent enflurane (20 patients), or fentanyl (20 patients). Fentanyl was administered iv as a 100–150 μg loading dose and supplemented with 50 μg every 15 min until approximately 30 min before the anticipated completion of operation. Each anesthetic group consisted of ten patients undergoing cholecystectomy or hysterectomy. Patients were randomly assigned to anesthetic groups, with the exception of those receiving halothane for cholecystectomy. These patients were not studied until completion of the other study groups and, therefore, represent ten consecutive patients. Pancuronium was administered to all patients to provide intraoperative muscle relaxation. Intravenous fluid administration consisted of 5–8 ml/kg/hr lactated Ringer's solution. Blood replacement was not necessary in any patient.

Samples of venous blood for LDH_5 determinations were obtained the evening before operation (control) and then one and 24 hours postoperatively. Results were analyzed by analysis of variance, and $P < 0.05$ was considered significant.

* Assistant Professor.
† Professor and Chairman.
Received from the Department of Anesthesia, Indiana University School of Medicine, Indianapolis, Indiana 46223. Accepted for publication May 1, 1979.
Address reprint requests to Dr. Viegas.

0003-3022/79/1200/0556 $00.60

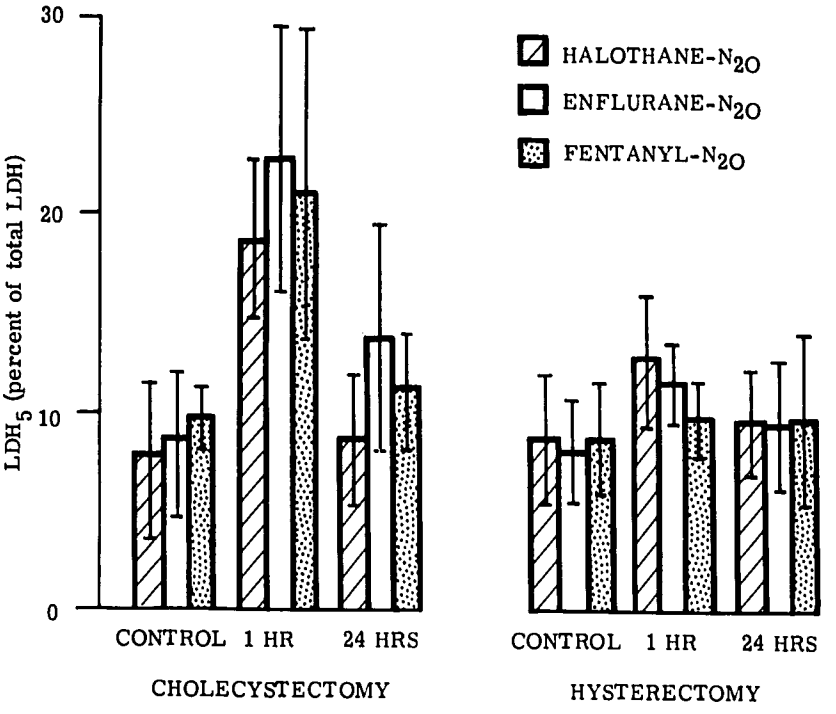

FIG. 1. LDH_5 (per cent of total LDH, mean ± SD) before and one and 24 hours after elective cholecystectomy (30 patients) or hysterectomy (30 patients) in patients anesthetized with 60 per cent inspired nitrous oxide plus halothane, enflurane, or fentanyl. Each anesthetic group included ten patients. Normal LDH_5 is 5–16 per cent of total LDH.

LDH_5 did not change following hysterectomies. In contrast, LDH_5 values one hour after cholecystectomies were increased above control ($P < 0.05$) and above corresponding values ($P < 0.05$) in those patients undergoing hysterectomy. These LDH_5 changes were not influenced by the anesthetic drugs. LDH_5 values 24 hours after cholecystectomies were not different from control or corresponding measurements after hysterectomies.

RESULTS

Patient ages, weights, and sex distribution and durations of operations are summarized in table 1.

Control LDH_5 values (normal 5–16 per cent of total LDH) were similar in all groups (fig. 1). One hour after operation, LDH_5 was increased ($P < 0.05$) above control only in patients undergoing cholecystectomy. These one hour measurements were significantly greater than corresponding measurements following hysterectomy. The magnitudes of increase after cholecystectomy were similar for all three anesthetic drug combinations. Twenty-four hours after cholecystectomy, LDH_5 values had decreased and were no longer significantly different from control values or corresponding values following hysterectomy.

LDH_5 changes in individual patients did not reveal any exaggerated response that would have been masked by pooling data. Twenty-four hours after hysterectomy only one patient, anesthetized with fentanyl–N_2O, had an LDH_5 above normal (22 per cent). Twenty-four hours after cholecystectomy LDH_5 was greater than 16 per cent in only four patients (17, 20, 22, and 26 per cent). All these patients had received enflurane–N_2O.

DISCUSSION

The two variables in this study that seemed most likely to contribute to hepatic dysfunction postoperatively were the anesthetic drug (halothane, enflurane, or fentanyl) and the site of operation (upper or lower abdominal). Other factors that may contribute to postoperative hepatic dysfunction, such as pre-existing hepatic disease, nutritional status, repeated operation, blood transfusion, sepsis, hypotension, and duration of operation, were either absent or similarly present in all study groups.

These data demonstrate that LDH_5 changes postoperatively were similar regardless of the anesthetic drug. The greater LDH_5 increases following cholecystectomies as compared with hysterectomies are in agreement with previous reports demonstrating that proximity of the surgical field to the liver rather than the specific anesthetic drug is the important determinant of postoperative hepatocellular dysfunction.[4,5] Our data do not support an earlier report that halothane anesthesia for cholecystectomy was associated with greater and more prolonged LDH_5 elevations than those occurring after anesthesia not including halothane.[2]

A much larger series is necessary to detect the rare

TABLE 1. Patient Data (Mean ± SD)

	Age (Years)	Weight (kg)	Sex	Duration of Operation (Min)
Cholecystectomy (30 patients)	40 ± 9	73 ± 10	F 22 M 8	105 ± 28*
Hysterectomy (30 patients)	47 ± 11	66 ± 12	F 30	145 ± 35

* $P < 0.05$, cholecystectomy versus hysterectomy.

patient manifesting anesthetic-associated hepatic dysfunction. The importance of the present data is to emphasize the absence of detectable differences between commonly used anesthetic drugs with respect to postoperative hepatic dysfunction. Indeed, evidence of hepatic dysfunction in our study, using a sensitive indicator of hepatocellular damage (LDH_5), was minimal even in those patients undergoing upper abdominal operations. For example, LDH_5 remained above normal in only four of 30 patients one day following cholecystectomy. Although these four patients all had received enflurane, the LDH_5 elevations were minimal, and we feel in this small series should not be interpreted as representing a specific anesthetic effect.

References

1. Batsakis JG, Siders D: Enzyme molecular heterogeneity (isoenzymes) in surgical diagnosis. Arch Surg 95:138–143, 1967
2. Klar H, Lundstrom J, Molleberg H, et al: The isoenzyme pattern of LDH following halothane anesthesia for cholecystectomy. Anaesthetist 23:417–420, 1974
3. Kalow B, Rogoman, E, Sims FH: A comparison of the effects of halothane and other anaesthetic agents on hepatocellular function in patients submitted to elective operations. Canad Anaesth Soc J 23:71–78, 1976
4. Clarke RSJ, Roggart JR, Lavery T: Changes in liver function after different types of surgery. Br J Anaesth 48:119–127, 1976
5. Hobson RW, Conant C, Fleming A, et al: Postoperative serum enzyme patterns. Milit Med 136:624–628, 1971

Anesthesiology
51:558–560, 1979

Bronchoscopy and Reversal of Intracardiac Shunt

TADIKONDA L. K. RAO, M.D.,* MALI MATHRU, M.D.,* C. AZAD, M.D.,† ADEL A. EL-ETR, M.D.,‡ M. R. SALEM, M.D.§

The direction of shunt is from left to right in patients with atrial septal defect, because of the higher left atrial pressure (LAP) compared with the right atrial pressure (RAP).[1] Various conditions, including pulmonary embolism, hypercarbia, acidemia, systemic arterial hypotension, and increased airway pressure, may reverse the shunt and result in arterial hypoxemia.[2,4] The following case demonstrates the reversal of a left-to-right shunt with the institution of positive-pressure breathing during bronchoscopy.

Report of a Case

A 65-year-old man was admitted with a history of hemoptysis and was scheduled for bronchoscopy. Past history revealed that since birth he had had a heart murmur. He had no limitation of activity, and no history of cyanotic spells. Cardiac catheterization performed during the present admission revealed an atrial septal defect with 4-to-1 left-to-right shunt, with normal valves, coronary arteries, and pressures. Results of other laboratory investigations were within normal limits. Preoperative blood-gas values during breathing of room air were: *p*H 7.42, P_{O_2} 91 torr, P_{CO_2} 36 torr, and base excess 2.

The patient was premedicated with atropine sulfate, 0.4 mg, and secobarbital, 100 mg, im, an hour prior to the anticipated bronchoscopy. In the operating room, ECG and heart rate were continuously monitored. Blood pressure was monitored by an appropriate-sized cuff. Prior to the induction of anesthesia, vital signs were: heart rate 84/min, blood pressure 126/88 torr, respiratory rate 16/min. ECG revealed normal sinus rhythm. After preoxygenation, anesthesia was induced with a sleep dose of thiopental and endotracheal intubation was facilitated with succinylcholine. Anesthesia was maintained with 50 per cent nitrous oxide in oxygen, supplemented by fentanyl, and muscular relaxation was maintained by infusion of 0.2 per cent succinylcholine. Ventilation was controlled manually, and breath sounds were satisfactory. An adult-sized flexible fiberoptic bronchoscope was passed through the endotracheal tube, and bronchoscopy revealed a small mass in the right main bronchus. Throughout this time, patient was manually ventilated, and vital signs were stable. It was decided to perform a rigid bronchoscopy to obtain a biopsy of the mass. The patient's trachea was extubated and a 7-mm rigid bronchoscope passed into the trachea without difficulty. A Sanders ventilating attachment was connected to the proximal end of the bronchoscope, and the patient was ventilated at a rate of 15/min using a mixture of 50 per cent nitrous oxide in oxygen delivered by a high-flow N_2O–O_2 blender, which delivered the gas mixture at a constant pressure of 50 psi. Vital signs were stable, and chest expansions were good and bilaterally equal with each jet inflation. Analysis of a sample of arterial blood obtained at this time revealed *p*H 7.44, P_{O_2} 120 torr, and P_{CO_2} 32 torr. After 10 min of bronchoscopy, the patient was noticed to be cyanotic and tachycardic, with a rate of 130/min. An arterial blood sample was drawn, and analysis revealed *p*H 7.38, P_{O_2} 49 torr, and P_{CO_2} 42 torr. The patient was ventilated with 100 per cent oxygen with the Sander's jet ventilation attachment through the bronchoscope. Since the cyanosis did not abate, another arterial blood sample was drawn, and analysis revealed *p*H 7.19, P_{O_2} 59 torr, P_{CO_2} 48 torr, and a base deficit of 7. The bronchoscope was withdrawn, the trachea was reintubated with 9-mm endotracheal tube, and manual ventilation was instituted with 100 per cent oxygen. Sodium bicarbonate, 50 mEq, was given iv to correct the metabolic acidosis. Five minutes later, since the cyanosis was still present, another

* Assistant Professor.
† Fellow in Cardiovascular Anesthesia.
‡ Professor and Chairman.
§ Professor and Assistant Chairman.

Received from the Department of Anesthesiology, Loyola University Stritch School of Medicine, 2160 South First Avenue, Maywood, Illinois 60153. Accepted for publication May 1, 1979.

Address reprint requests to Dr. Rao.

0003-3022/79/1200/0558 $00.65