

# The First Case Report of Local Anesthesia Reversal (LAR) of the Upper Arm Brachial Plexus Block by Lipid Emulsion

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## Abstract

A 25 years old male was diagnosed as having natural stump in left forearm due to detonator. He needed an emergency surgery for cutting damaged tissue and repair of the stump. The patient was anesthetized by Ultrasound guided brachial plexus block through the Supra Clavicular approach with Lidocaine 4 mg/kg combined with Ropivacaine 1mg/kg and Epinephrine 1/200.000 in total of 30 ml solution. Lipid emulsion (Lipofundin 20%, B. Braun) bolus injection was given 70 min after the Brachial plexus block over 2 min in a dose of 1.5 mg/kg (patient's weight is 60 kg). After 2 min of the Lipid emulsion bolus injection he could move slightly his left arm. Full Local Anesthesia Reversal (LAR) was achieved after 255 min from the Brachial plexus block performance.

It is the first case report in the medical literature of Local Anesthesia Reversal (LAR) of the upper arm Brachial plexus block by Lipid Emulsion.

**Keywords:** Intralipid, Lipid emulsion, LE, Lipofundin, Ropivacaine supraclavicular block, Brachial plexus block, Local anesthesia reversal, LAR.

## Case report

A 25 years old male was diagnosed as having natural stump in left forearm due to detonator.

(Photo: <http://www.csen.com/stump1.jpg>)

(X-ray: <http://www.csen.com/stump2.jpg>)

He needed an emergency surgery for cutting damaged tissue and repair of the stump. The operation was done on Feb 16th, 2018 at the Military Hospital 103, Vietnam Military Medical University. The patient was anesthetized by Ultrasound guided brachial plexus block through the Supra Clavicular approach with Lidocaine 4 mg/kg combined with Ropivacaine 1mg/kg and Epinephrine 1/200.000 in total of 30 ml solution.

- Onset time of Anesthesia: 5 min.
- Anesthesia effect was very good for the surgery.
- Duration of surgery: 50 min

Surgery was accomplished in 65 min after the Brachial plexus block. Sensory and motor function of the left hand that were assessed before injection were absent completely.

Lipid emulsion (Lipofundin 20%, B. Braun) bolus injection was given 70 min after the Brachial plexus block over 2 min in a dose of 1.5 mg/kg (patient's weight is 60 kg). The patient was monitored closely in the operating room for 1 hour:

## Article Information

**Article Type:** Research

**Article Number:** JHSD108

**Received Date:** 27 February, 2018

**Accepted Date:** 12 March, 2018

**Published Date:** 15 March, 2018

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**Citation:** Joseph Eldor, Hien VV, Kien NT (2018) The First Case Report of Local Anesthesia Reversal (LAR) of the Upper Arm Brachial Plexus Block by Lipid Emulsion. J Health Sci Dev Vol: 1, Issu: 1 (61-66).

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(Video 1: [https://www.youtube.com/watch?v=93QrTB-2\\_o](https://www.youtube.com/watch?v=93QrTB-2_o))

After 2 min of the Lipid emulsion bolus injection he could move slightly his left arm. Vital signs were stable after the Lipid emulsion bolus injection.

After 185 min from the lipid emulsion bolus injection the sensory and motor function of the left hand were fully recovered. He could move his left hand without any difficulty. It means that full Local Anesthesia Reversal (LAR) was achieved after 255 min from the Brachial plexus block performance.

(Video 2: <https://www.youtube.com/watch?v=K2H4h-GLZko&feature=youtu.be>)

## Discussion

### Supraclavicular block

The first percutaneous supraclavicular block was performed in 1911 by German surgeon Diedrich Kulenkampff (1880–1967)[1]. Kulenkampff subjected himself to the supraclavicular block. Later that year, Georg Hirschel (1875–1963) described a percutaneous approach to the brachial plexus from the axilla[2]. By the late 1940s, clinical experience with brachial plexus block in both peacetime and wartime surgery was extensive, and new approaches to this technique began to be described[3]. For example, In 1946, F. Paul Ansbros was the first to describe a continuous brachial plexus block technique. He secured a needle in the supraclavicular fossa and attached tubing connected to a syringe through which he could inject incremental doses of local anesthetic[4]. The subclavian perivascular block was first described by Winnie and Collins in 1964[5]. This approach became popular due to its lower risk of pneumothorax compared to the traditional Kulenkampff approach. The infraclavicular approach was first developed by Raj. In 1977, Selander described a technique for continuous brachial plexus block using an intravenous catheter secured in the axilla[6].

### Prolonged blockade

There are a few reports of prolonged blockade following seemingly flawless technique of performing block. Complete recovery in those case reports varied from 40 to 84 hours after the block[7-8]. None of the papers have clearly stated the reason behind the long blockade. Injecting the local anesthetic too close to the nerves and chronic treatment with lithium has been proposed as reasons behind these unusually prolonged blocks. Ludueña believed that causes of prolonged blockade are often unknown and if the duration is longer than 24 hours then probability of nerve damage should be considered[9].

### Regular blockade

The present study compares the effectiveness of 0.25% ropivacaine and 0.25% bupivacaine in 44 patients receiving a subclavian perivascular brachial plexus block for upper extremity surgery. The patients were assigned to two equal groups in this randomized, double-blind study; one group

received ropivacaine 0.25% (112.5 mg) and the other, bupivacaine 0.25% (112.5 mg), both without epinephrine. Onset times for analgesia and anesthesia in each of the C-5 through T-1 brachial plexus dermatomes did not differ significantly between the two groups. The mean onset time for analgesia ranged from 11.2 to 20.2 min, and the mean onset time for anesthesia ranged from 23.3 to 48.2 min. The onset of motor block differed only with respect to paresis in the hand, with bupivacaine demonstrating a shorter onset time than ropivacaine. The duration of sensory and motor block also was not significantly different between the two groups. The mean duration of analgesia ranged from 9.2 to 13.0 h, and the mean duration of anesthesia ranged from 5.0 to 10.2 h. Both groups required supplementation with peripheral nerve blocks or general anesthesia in a large number of cases, with 9 of the 22 patients in the bupivacaine group and 8 of the 22 patients in the ropivacaine group requiring supplementation to allow surgery to begin. In view of the frequent need for supplementation noted with both 0.25% ropivacaine and 0.25% bupivacaine, we do not recommend using the 0.25% concentrations of these local anesthetics to provide brachial plexus block [10].

The effects of clonidine and epinephrine, administered into the brachial plexus sheath, were evaluated in 60 patients who underwent surgery of the upper limb. All patients received 40 to 50 ml of 0.25% bupivacaine, injected into the brachial plexus sheath, using the supraclavicular technique. The patients were randomly allocated to two groups so that 30 patients received 150 micrograms clonidine hydrochloride (Group I), and 30 received 200 micrograms epinephrine (Group II). The quality and the duration of analgesia were assessed as well as the possible side-effects. The block produced with the addition of clonidine was longer (994.2 +/- 34.2 vs 728.3 +/- 35.8 min) and superior to that with epinephrine (P less than 0.001). No major side-effects were recorded. We conclude that the injection of clonidine into the brachial plexus sheath is an attractive alternative to epinephrine to prolong the duration of analgesia following upper limb surgery under conduction anaesthesia[11].

This study compared the effectiveness of 0.5% ropivacaine and 0.5% bupivacaine for brachial plexus block[12]. Forty-eight patients received a subclavian perivascular brachial plexus block for upper-extremity surgery. One group (n=24) received ropivacaine 0.5% (175 mg) and a second group (n=24) received bupivacaine 0.5% (175 mg), both without epinephrine. Onset times for analgesia and anesthesia in each of the C5 through T1 brachial plexus dermatomes did not differ significantly between groups. Duration of analgesia and anesthesia was long (mean duration of analgesia, 13-14h; mean duration of anesthesia, 9-11h) and also did not differ significantly between groups. Motor block was profound, with shoulder paralysis as well as hand paresis developing in all of the patients in both groups. Two patients in each group required supplemental blocks before surgery. Ropivacaine 0.5% and bupivacaine 0.5% appeared equally effective in providing brachial plexus anesthesia[12].

The mixture of 1% lidocaine and 0.2% tetracaine with 1:200,000 epinephrine, so-called "superacaine," has been

used extensively for axillary brachial plexus blockade for several decades. Since the advent of bupivacaine, the supercaine mixture has fallen into relative disuse despite its record of effectiveness and safety. No studies have been done recently to evaluate quality of anesthesia, duration of postoperative analgesia, and degree of patient satisfaction with this mixture when used for axillary brachial plexus blockade. The assumptions were as follows: surgical anesthesia will be adequate, length of postoperative analgesia will be approximately 4 to 9 hours, and patients will be highly satisfied. The specific aim of the present study was to describe the anesthetic characteristics of supercaine. Patients between 18 and 65 years of age received a standard mixture of supercaine, totalling 450-500 mg of lidocaine and 90 to 100 mg of tetracaine. Epinephrine in a solution of 1:200,000 and an 8.4% solution of sodium bicarbonate were added, and the trans arterial technique was used. Patients were contacted on postoperative day 1 to determine the duration of sensory and motor block; overall satisfaction with the block was rated. Data were analyzed with the Statistical Program for the Social Sciences (SPSS, Chicago, Ill) and Stata (Stata Corp., College Station, Tex) computer programs. The mean +/- SD findings were as follows: duration of sensory block, 465 +/- 204 minutes; duration of motor block, 473 +/- 214 minutes; patient satisfaction score, 9 +/- 1 on a 1 to 10 scale. Data are reported within a 95% confidence interval. Variables examined and compared were not statistically significant. We concluded that the duration of block supports findings reported in the literature, patients equate duration of sensory block with duration of motor block, differences in duration were probably due to levels of provider experience, and patients were extremely satisfied with the anesthetic[13].

Ultrasound-guided supraclavicular brachial plexus block (USSB) provides excellent postoperative analgesia after upper extremity surgery. Dexamethasone and clonidine have been added to local anesthetics to enhance and prolong the duration of analgesia.

The objective of this randomized prospective study is to evaluate the efficacy of dexamethasone, clonidine, or combination of both as adjuvants to ropivacaine on the duration of USSB for postoperative analgesia.

Patients receiving USSB for postoperative pain control for upper extremity surgery were randomized to one of four groups; ropivacaine 0.5 %, ropivacaine 0.5 % with 4 mg dexamethasone, ropivacaine 0.5% with 100 mcg clonidine , or ropivacaine 0.5 % with 4 mg dexamethasone and 100 mcg clonidine. Pain scores, sensory and motor function were evaluated at post anesthesia care unit (PACU), discharge and at 24 h postoperatively.

The duration of sensory and motor blocks were significantly longer in clonidine groups when compared to ropivacaine alone [Sensorial analgesia: ropivacaine alone 13.4±6, Ropivacaine-Clonidine 17.4 ± 6; Ropivacaine-Dexamethasone-Clonidine 18.8±6.2; Motor blocks: Ropivacaine 12 ± 5, Ropivacaine-Clonidine 16.8 ± 5.2, Ropivacaine-Dexamethasone-Clonidine 18.2 ± 5.7]. In clonidine groups, there was significantly prolongation of

motor and sensory block when compared to ropivacaine alone group.

The results demonstrated that clonidine significantly prolongs the duration of ropivacaine effects for the postoperative analgesia in patient underwent upper arm surgeries[14].

The duration of effect for axillary plexus block using ropivacaine is highly variable. The available literature does not offer any plausible means of predicting time of block offset for individual patients, making it difficult to give accurate information and plan postoperative analgesics. This study was designed to identify factors influencing axillary plexus block offset time.

A total of 92 patients participated in this prospective double centred observational study. All patients were scheduled for axillary plexus block with ropivacaine 0.75% and subsequent block duration was recorded.

Mean time of axillary plexus block offset was 13.5 hours, with a range of 4.8 to 25.4 hours. No statistical significant differences in offset time was seen with regard to gender, age, body weight, BMI and ASA-classification. A trend for increasing duration of blocks associated with increasing age was observed. No statistically significant difference was identified in block duration between blocks performed with nerve stimulator guidance versus ultrasound guidance. Similarly, neither dose nor volume of ropivacaine 0.75% was identified as a factor influencing block duration.

This prospective study demonstrates a large inter individual variation in time of axillary plexus block offset using ropivacaine 0.75%. The lack of association between offset time and both demographic and block performance factors, makes predictability of individual duration of axillary plexus blocks in clinical practice extremely difficult. We suggest that all patients should be made aware of such variability in duration prior to block placement[15].

On a pharmacologic basis, levobupivacaine is expected to last longer than ropivacaine. However, most reports of these anesthetics for brachial plexus block do not suggest a difference in analgesic effect. The aim of this study is to compare the postoperative analgesic effects of levobupivacaine and ropivacaine when used for treating ultrasound-guided brachial plexus block.

A total of 62 patients undergoing orthopaedic surgery procedures were prospectively enrolled and randomized to receive levobupivacaine (group L, N=31) or ropivacaine (group R, N=31). The duration of analgesia, offset time of motor block, need for rescue analgesics, and sleep disturbance on the night of surgery were recorded. Pain score was recorded on the day of surgery, and on postoperative days 1 and 2.

There was no difference in the time interval until the first request for pain medication comparing the two groups (group L: 15.6 [11.4, 16.8] hours; group R: 12.5 [9.4, 16.0] hours, P=0.32). There was no difference in the duration of motor block (group L: 12.2 [7.6, 14.4] hours; group R: 9.4 [7.9, 13.2] hours, P=0.44), pain score (P=0.92), need for

rescue analgesics (group L: 55%; group R: 65%,  $P=0.6$ ), or rate of sleep disturbance (group L: 61%, group R: 58%,  $P=1.0$ ) on comparing the two groups.

There was no difference in postoperative analgesia comparing levobupivacaine and ropivacaine when used for brachial plexus block[16].

For any surgery in the upper extremity that does not involve the shoulder, a supraclavicular block is preferred, as it is a safe procedure associated with rapid onset and reliable anaesthesia. Although ropivacaine has been extensively studied for epidural anaesthesia, very few reports exist on its use in supraclavicular brachial plexus block.

This study was conducted to investigate and compare the effectiveness of supraclavicular brachial plexus anaesthesia with two different concentrations of ropivacaine (0.5% and 0.75%) and to compare them with the standard 0.5% bupivacaine[17].

Ninety patients of age 18 to 60 years belonging to American Society of Anaesthesiologists (ASA) status 1 or 2, admitted to Pondicherry Institute of Medical Sciences were chosen for the study and were divided into three groups. Group A received 30 ml of 0.5% bupivacaine, group B received 30 ml of 0.5% ropivacaine and group C received 30 ml of 0.75% ropivacaine into the supraclavicular region, by a nerve-stimulator technique. Onset time of each of the drug was recorded both for the sensory and motor block. Duration of sensory and motor block was recorded along with peri-operative haemodynamic monitoring.

The onset of complete sensory and motor block observed with both ropivacaine groups and bupivacaine was similar ( $16.85\pm 6.67$  min in group A,  $17.79\pm 5.03$  min in group B and  $18.48\pm 6.14$  in group C,  $p>0.05$ ); onset of motor block ( $21.45\pm 4.45$  min in group A,  $22.23\pm 4.05$  min in group B and  $22.33\pm 5.17$  in group C,  $p<0.05$ ). The duration of sensory block with 0.5% bupivacaine was 11.58 hours, with 0.5% ropivacaine was 9.02 hours with 0.75% ropivacaine was 8.87 hours ( $p<0.001$ ). The duration of motor block with 0.5% bupivacaine was 12.94 hours, with 0.5% ropivacaine was 8.29 hours with 0.75% ropivacaine was 7.89 hours ( $p<0.001$ ). Multiple comparison test with Bonferroni correction showed there was statistically significant difference in mean duration of sensory block between Group A (0.5% bupivacaine) and Group B (0.5% ropivacaine) and also between Group A (0.5% bupivacaine) and Group C (0.75% ropivacaine). However, there were no statistically significant difference in mean duration of sensory block between Group B (0.5% ropivacaine) and Group C (0.75% ropivacaine). The preoperative, intra operative and postoperative heart rate, systolic & diastolic blood pressure and oxygen saturation were comparable among the three study groups ( $p>0.05$ ). No side effects were recorded in the study.

The onset of sensory and motor block was similar in all the three groups. However, when compared to bupivacaine group, recovery of motor functions was faster in both the ropivacaine groups. Patients in all the 3 groups did not experience any adverse effects[17].

## Lipid Emulsion Effects on Mitochondria and Intracellular Calcium

Local anesthetic toxicity is thought to be mediated partly by inhibition of cardiac mitochondrial function. Intravenous (i.v.) lipid emulsion may overcome this energy depletion, but doses larger than currently recommended may be needed for rescue effect. In this randomized study with anesthetized pigs, we compared the effect of a large dose, 4 mL/kg, of i.v. 20% Intralipid® (n=7) with Ringer's acetate (n=6) on cardiovascular recovery after a cardiotoxic dose of bupivacaine[18]. We also examined mitochondrial respiratory function in myocardial cell homogenates analyzed promptly after needle biopsies from the animals. Bupivacaine plasma concentrations were quantified from plasma samples. Arterial blood pressure recovered faster and systemic vascular resistance rose more rapidly after Intralipid than Ringer's acetate administration ( $p<0.0001$ ), but Intralipid did not increase cardiac index or left ventricular ejection fraction. The lipid-based mitochondrial respiration was stimulated by approximately 30% after Intralipid ( $p<0.05$ ) but unaffected by Ringer's acetate. The mean (standard deviation) area under the concentration-time curve (AUC) of total bupivacaine was greater after Intralipid (105.2 (13.6) mg·min/L) than after Ringer's acetate (88.1 (7.1) mg·min/L) ( $p=0.019$ ). After Intralipid, the AUC of the lipid-un-entrapped bupivacaine portion (97.0 (14.5) mg·min/L) was 8% lower than that of total bupivacaine ( $p<0.0001$ ). To conclude, 4 mL/kg of Intralipid expedited cardiovascular recovery from bupivacaine cardiotoxicity mainly by increasing systemic vascular resistance. The increased myocardial mitochondrial respiration and bupivacaine entrapment after Intralipid did not improve cardiac function[18].

Lipid emulsions have been used to treat various drug toxicities and for total parenteral nutrition therapy. Their usefulness has also been confirmed in patients with local anesthetic-induced cardiac toxicity. The purpose of this study was to measure the hemodynamic and composition effects of lipid emulsions and to elucidate the mechanism associated with changes in intracellular calcium levels in myocytes.

We measured hemodynamic effects using a digital analysis system after Intralipid® and Lipofundin® MCT/LCT were infused into hearts hanging in a Langendorff perfusion system[20]. We measured the effects of the lipid emulsions on intracellular calcium levels in H9c2 cells by confocal microscopy.

Infusion of Lipofundin® MCT/LCT 20% (1ml/kg) resulted in a significant increase in left ventricular systolic pressure compared to that after infusing modified Krebs-Henseleit solution (1ml/kg) ( $P=0.003$ , 95% confidence interval [CI], 2.4-12.5). Lipofundin® MCT/LCT 20% had a more positive inotropic effect than that of Intralipid® 20% ( $P=0.009$ , 95% CI, 1.4-11.6). Both lipid emulsion treatments increased intracellular calcium levels. Lipofundin® MCT/LCT (0.01%) increased intracellular calcium level more than that of 0.01% Intralipid® ( $P<0.05$ , 95% CI, 0.0-1.9).

These two lipid emulsions had different inotropic effects depending on their triglyceride component. The inotropic effect of lipid emulsions could be related with intracellular calcium level[19].

Accidental intravascular or high-dose injection of local anesthetics (LA) can result in serious, potentially life-threatening complications. Indeed, adequate supportive measures and the administration of lipid emulsions are required in such complications. The study's objectives were threefold: (i) evaluate the myocardial toxicity of levobupivacaine when administered intravenously; (ii) investigate levobupivacaine toxicity on cardiomyocytes mitochondrial functions and cellular structure; (iii) assess the protective effects of a lipid emulsion in the presence or absence of myocardial ischemia. Domestic pigs randomized into two groups of 24 animals each, with either preserved coronary circulation or experimental myocardial ischemia. Six animals from each group received either: (i) single IV injection of saline, (ii) lipid emulsion (Intralipid®), (iii) levobupivacaine, (iv) combination levobupivacaine-Intralipid®. Serially measured endpoints included: heart rate, duration of the monophasic action potentials (dMAP), mean arterial pressure, and peak of the time derivative of left ventricular pressure (LV dP/dtmax). In addition, the following cardiomyocytes mitochondrial functions were measured: reactive oxygen species (ROS) production, oxidative phosphorylation, and calcium retention capacity (CRC) as well as the consequences of ROS production on lipids, proteins, and DNA. IV injection of levobupivacaine induced sinus bradycardia and reduced dMAP and LV dP/dtmax. At the mitochondrial level, oxygen consumption and CRC were decreased. In contrast, ROS production was increased leading to enhanced lipid peroxidation and structural alterations of proteins and DNA. Myocardial ischemia was associated with global worsening of all changes. Intralipid® quickly improved haemodynamics. However, beneficial effects of Intralipid® were less clear after myocardial ischemia[20].

Cocaine intoxication leads to over 500,000 emergency department visits annually in the United States and ethanol cointoxication occurs in 34% of those cases. Cardiotoxicity is an ominous complication of cocaine and cocaethylene overdose for which no specific antidote exists. Because infusion of lipid emulsion (Intralipid) can treat lipophilic local anesthetic toxicity and cocaine is an amphipathic local anesthetic, the author tested whether lipid emulsion could attenuate cocaine cardiotoxicity *in vivo*[21]. The effects of lipid emulsion were compared with the metabolically inert sulfobutylether- $\beta$ -cyclodextrin (SBE- $\beta$ -CD; Captisol) in an isolated heart model of cocaine and cocaethylene toxicity to determine if capture alone could exert similar benefit as lipid emulsion, which exhibits multimodal effects. The authors then tested if cocaine and cocaethylene, like bupivacaine, inhibit lipid-based metabolism in isolated cardiac mitochondria.

For whole animal experiments, Sprague-Dawley rats were anesthetized, instrumented, and pretreated with lipid emulsion followed by a continuous infusion of cocaine

to assess time of onset of cocaine toxicity. For *ex vivo* experiments, rat hearts were placed onto a nonrecirculating Langendorff system perfused with Krebs-Henseleit solution. Heart rate, left ventricle maximum developed pressure (LVdevP), left ventricle diastolic pressure, maximum rate of contraction (+dP/dtmax), maximum rate of relaxation (-dP/dtmax), rate-pressure product (RPP = heart rate $\times$ LVdevP), and line pressure were monitored continuously during the experiment. A dose response to cocaine (10, 30, 50, and 100  $\mu$ mol/L) and cocaethylene (10, 30, and 50  $\mu$ mol/L) was generated in the absence or presence of either 0.25% lipid emulsion or SBE- $\beta$ -CD. Substrate-specific rates of oxygen consumption were measured in interfibrillar cardiac mitochondria in the presence of cocaine, cocaethylene, ecgonine, and benzoylecgonine.

Treatment with lipid emulsion delayed onset of hypotension (140 seconds vs. 279 seconds;  $p=0.008$ ) and asystole (369 seconds vs. 607 seconds;  $p=0.02$ ) in whole animals. Cocaine and cocaethylene induced dose-dependent decreases in RPP, +dP/dtmax, and -dP/dtmaxabs ( $p<0.0001$ ) in Langendorff hearts; line pressure was increased by cocaine and cocaethylene infusion, but not altered by treatment. Lipid emulsion attenuated cocaine- and cocaethylene-induced cardiac depression. SBE- $\beta$ -CD alone evoked a mild cardiodepressant effect ( $p<0.0001$ ) but attenuated further cocaine- and cocaethylene-induced decrements in cardiac contractility at high concentrations of drug (100  $\mu$ mol/L;  $p<0.001$ ). Finally, both cocaine and cocaethylene, but not ecgonine and benzoylecgonine, inhibited lipid-dependent mitochondrial respiration by blocking carnitine exchange ( $p<0.05$ ). A commercially available lipid emulsion was able to delay progression of cocaine cardiac toxicity *in vivo*. Further, it improved acute cocaine- and cocaethylene-induced cardiac toxicity in rat isolated heart while SBE- $\beta$ -CD was effective only at the highest cocaine concentration. Further, both cocaine and cocaethylene inhibited lipid-dependent mitochondrial respiration. Collectively, this suggests that scavenging-independent effects of lipid emulsion may contribute to reversal of acute cocaine and cocaethylene cardiotoxicity, and the beneficial effects may involve mitochondrial lipid processing[21].

We hypothesized that acute lipid-induced insulin resistance would be attenuated in high-oxidative muscle of lean trained (LT) endurance athletes due to their enhanced metabolic flexibility and mitochondrial capacity[22]. Lean sedentary (LS), obese sedentary (OS), and LT participants completed two hyperinsulinemic euglycemic clamp studies with and without (glycerol control) the coinfusion of Intralipid. Metabolic flexibility was measured by indirect calorimetry as the oxidation of fatty acids and glucose during fasted and insulin-stimulated conditions, the latter with and without lipid oversupply. Muscle biopsies were obtained for mitochondrial and insulin-signaling studies. During hyperinsulinemia without lipid, glucose infusion rate (GIR) was lowest in OS due to lower rates of nonoxidative glucose disposal (NOGD), whereas state 4 respiration was increased in all groups. Lipid infusion reduced GIR similarly in all subjects and reduced state 4 respiration. However, in LT subjects, fat oxidation was higher with lipid oversupply, and

although glucose oxidation was reduced, NOGD was better preserved compared with LS and OS subjects. Mitochondrial performance was positively associated with better NOGD and insulin sensitivity in both conditions. We conclude that enhanced mitochondrial performance with exercise is related to better metabolic flexibility and insulin sensitivity in response to lipid overload[22].

## Conclusion

It is the first case report in the medical literature of Local Anesthesia Reversal (LAR) of the upper arm Brachial plexus block by Lipid Emulsion.

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