Our conclusion is in agreement with the ultracentrifugal and ethyl alcohol fractionation findings of Graham *et al.*[3].

The heparin used in our experiments was kindly supplied by Boots Pure Drug Co., Ltd.

A. S. Achimastos
A. N. Granitsas

Institute of Physiology,
University of Athens, Athens 609.

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Capillary Blood Standard Bicarbonate Values of Conscious and Anæsthetized Individuals

The clinical significance of blood standard bicarbonate has not yet been firmly established. Relatively little information is available at present concerning the effect of surgical anæsthesia on this new parameter in capillary or arterial blood. Payne and Conway[1] recently concluded that normal values for arterial blood standard bicarbonate were significantly lower than those reported by Astrup *et al.*[2] on capillary specimens. The latter workers contend that comparable standard bicarbonate values are obtained from arterial and capillary blood. Evaluation of acid-base balance during anæsthesia in our hospital revealed that standard bicarbonate concentrations of capillary blood from conscious and anæsthetized subjects were consistent with those of Astrup *et al.*[2].

Capillary blood collected from 15 clinically healthy medical students, analysed in Astrup equipment, yielded an average standard bicarbonate of 23·4 ± 1·6 m. equiv./l. The value is close to the mean (22·9 ± 1·5 m. equiv./l.) quoted by Astrup *et al.*[2] for capillary blood of 65 apparently normal persons. In line with these findings, Robinson[3] observed average standard bicarbonate-levels of 24·4 m. equiv./l. in arterialized venous blood of 20 fit males. Likewise, Papadopoulos and Keats[4] noted a mean arterial standard bicarbonate-level of 24·7 ± 0·7 m. equiv./l. in 20 subjects prior to orthopædic or gynæcological surgery.

A total of 124 standard bicarbonate estimations were made on capillary blood taken from 24 patients before, during and following major surgery. Anæsthesia was maintained by orotracheal administration of *cyclo*propane, nitrous oxide or halothane, following induction with thiopental and succinylcholine. Ventilation was controlled both by manual and mechanical methods. Standard bicarbonate-levels during anæsthesia ranged from 18·5 to 28·5 (mean 22·9 ± 2·3 m. equiv./l.). Although a majority of values fell within normal limits reported by Astrup *et al.*[2], 20 were above 24·8 m. equiv./l. and 8 dropped below 21·3 m. equiv./l. In anæsthetized patients, Robinson[3] as well as Papadopoulos and Keats[4] observed means of 23·3 and 21·8 m. equiv./l., respectively. The latter figure obtained on arterial blood tends to approach the lower standard bicarbonate-level (18·14 ± 1·94 m. equiv./l.) found by Payne and Conway[1] also in arterial blood of 22 patients anæsthetized with halothane. Individuals studied by Papadopoulos and Keats[4], however, were subjected to prolonged hyperventilation during surgical anæsthesia. Available evidence indicates that a modest to moderate compensatory metabolic acidosis frequently accompanies respiratory alkalosis[5].

Capillary blood collected from 16 patients during the first 2 hours following surgery yielded a mean standard bicarbonate of 23·6 ± 2·2 m. equiv./l. (range 18·6–26·5). These data compare favourably with the average obtained on medical students. A similar relationship between control and post-operative standard bicarbonate-levels was noted by Payne and Conway[1]. A reasonable explanation for the low values reported by these workers may stem from the use of arterial rather than capillary specimens. Ideally, arterial blood should be used for acid-base evaluation, but the collection procedure is not without danger to the patient. Woolmer[6] suggests that under general anæsthesia there is sufficient vasodilatation to effectively arterialize capillary blood.

R. L. Searcy
R. G. Craig
G. F. Gordon

Departments of Pathology and Anæsthesiology,
Los Angeles County General Hospital (Unit 2),
California College of Medicine, Los Angeles.

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Effect of Potassium Depletion on Lactic Dehydrogenase Activity in the Rat Nephron

In potassium depletion, the kidney is unable to concentrate the urine properly[1,2]. Striking morphological changes have been found in the region of the medulla and papilla[3,4,5]. In addition, marked changes in enzymatic activity, located mainly in the medulla, have been demonstrated by means of histochemical staining methods[6]. In the work reported here, lactic dehydrogenase activity was measured in various functional units of the kidney of normal and potassium-depleted rats through the use of the quantitative histochemical techniques originated by Lowry[7].

Male Sprague-Dawley rats, weighing between 100 and 150 g, were depleted of potassium over a 4-week period by feeding them a virtually potassium-free diet which contained a sodium polystyrene sulphonate resin and supplements of vitamins and minerals[8]. They drank a solution of 0·45 per cent sodium chloride and 0·45 per cent sodium bicarbonate. The test and control animals were pair fed. The controls were not given resin, while potassium chloride (50 m.equiv./l.) was added to the drinking water. Within three weeks the rats on the depletion diet became lethargic, lost weight, and developed a shaggy coat[8]. The mean serum potassium-level was decreased significantly from 5·7 m.equiv./l. (*S.E.* 0.41) for the control animals to 2·9 m.equiv./l. (*S.E.* 0·32) for the potassium-depleted group (P = 0·00027). After urine was collected on sheets of sterile aluminium foil, the animals were anesthetized with ether, the abdomen was opened, and blood was drawn from the inferior vena cava for chemical analysis. A 2-mm. thick cross-section was cut from the right kidney, extending from the renal capsule to the papilla tip. It was mounted on a microtome sample holder and quickly frozen in liquid nitrogen. The tissue was sectioned at 16μ in a cryostat at −20° C, lyophilized and used for microdissection, as described previously[9,10]. The individual structural units of the nephron were

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