# Use of sodium bicarbonate to treat tricyclic antidepressant-induced arrhythmias in a patient with alkalosis

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**Sodium bicarbonate has been recommended for the treatment of arrhythmias induced by tricyclic antidepressants. It is unclear, however, whether this therapy is effective only in the presence of acidosis. A case is presented in which there was an immediate response to sodium bicarbonate in three episodes of ventricular tachycardia despite the presence of alkalosis on two of the three occasions. Given the poor response to conventional therapy of arrhythmias induced by tricyclic antidepressants the use of sodium bicarbonate may be reasonable even in the presence of alkalosis. However, in the presence of pre-existing respiratory or metabolic alkalosis, such therapy is not without risk, and it is suggested that it be reserved for life-threatening situations when the arrhythmia has failed to respond to hyperventilation or antiarrhythmics or both.**

**Le bicarbonate de sodium a été proposé comme traitement des arythmies provoquées par des antidépresseurs tricycliques. Il n'est toutefois pas certain que ce traitement soit efficace seulement en présence d'acidose. On décrit un cas dans lequel il y a eu une réponse immédiate au bicarbonate de sodium lors de trois épisodes de tachycardie ventriculaire en dépit de la présence d'alcalose en deux des trois occasions. Étant donné la réponse médiocre des arythmies provoquées par des antidépresseurs tricycliques aux traitements traditionnels, l'emploi de bicarbonate de sodium paraît raisonnable même en présence d'alcalose. Cependant, ce traitement n'est pas sans risque en présence d'alcalose respiratoire ou métabolique pré-existante, et on suggère qu'il soit réservé aux situations menaçant le pronostic vital quand l'arythmie n'a pas cédé à l'hyperventilation, aux antiarythmiques ou aux deux.**

Overdose of a tricyclic antidepressant is frequently associated with cardiac arrhythmias, hypotension and congestive heart failure. Recent reports[1,2] have confirmed earlier observations[3] that the safest and most effective therapy is with either sodium bicarbonate or hyperventilation. The mechanism of the beneficial effect is not clear, although a change in protein binding, resulting in a decrease in the amount of biologically active free drug, has been suggested as the most likely explanation.[3] Since hyperventilation results in a more rapid onset of intracellular and extracellular pH changes, its use in emergency situations has been preferred by some.[2] Others have suggested that either therapy may be effective only in patients who have acidosis.[4] The following case report demonstrates the potential value of bicarbonate therapy even in a patient with alkalosis due to hyperventilation.

## Case report

A 23-year-old woman presented to a small country hospital 90 minutes after ingesting 5.35 g of imipramine hydrochloride. She was treated with ipecac syrup and gastric lavage, but there was minimal return of tablet particles.

She became comatose and hypotensive 1 hour after admission and was given 100 mL of 20% albumin. She was then transferred to our hospital's emergency department, arriving 3½ hours after admission to the first hospital; en route she had two major generalized seizures.

On admission to our hospital the serum levels of imipramine and its metabolite desipramine were 545 and 310 ng/mL respectively. She was hypotensive (systolic blood pressure 90 mm Hg, diastolic pressure palpable), cyanosed and comatose, with nonpurposeful response to deep pain. Her breathing was shallow, and an electrocardiogram (ECG) showed ventricular tachycardia (Fig. 1).

Intubation and hyperventilation with 100% oxygen were performed. The ventricular tachycardia persisted, however, and blood gas analysis showed metabolic acidosis with superimposed respiratory alkalosis (pH 7.36, partial pressures of oxygen and carbon dioxide in arterial blood 289 and 30 mm Hg respectively, and bicarbonate level 17 mmol/L). When 50 mmol of sodium bicarbonate was administered sinus rhythm appeared immediately and the pH rose to 7.52. Hyperventilation was continued, but ventricular tachycardia recurred after 5 minutes. The patient was given 2 mg of

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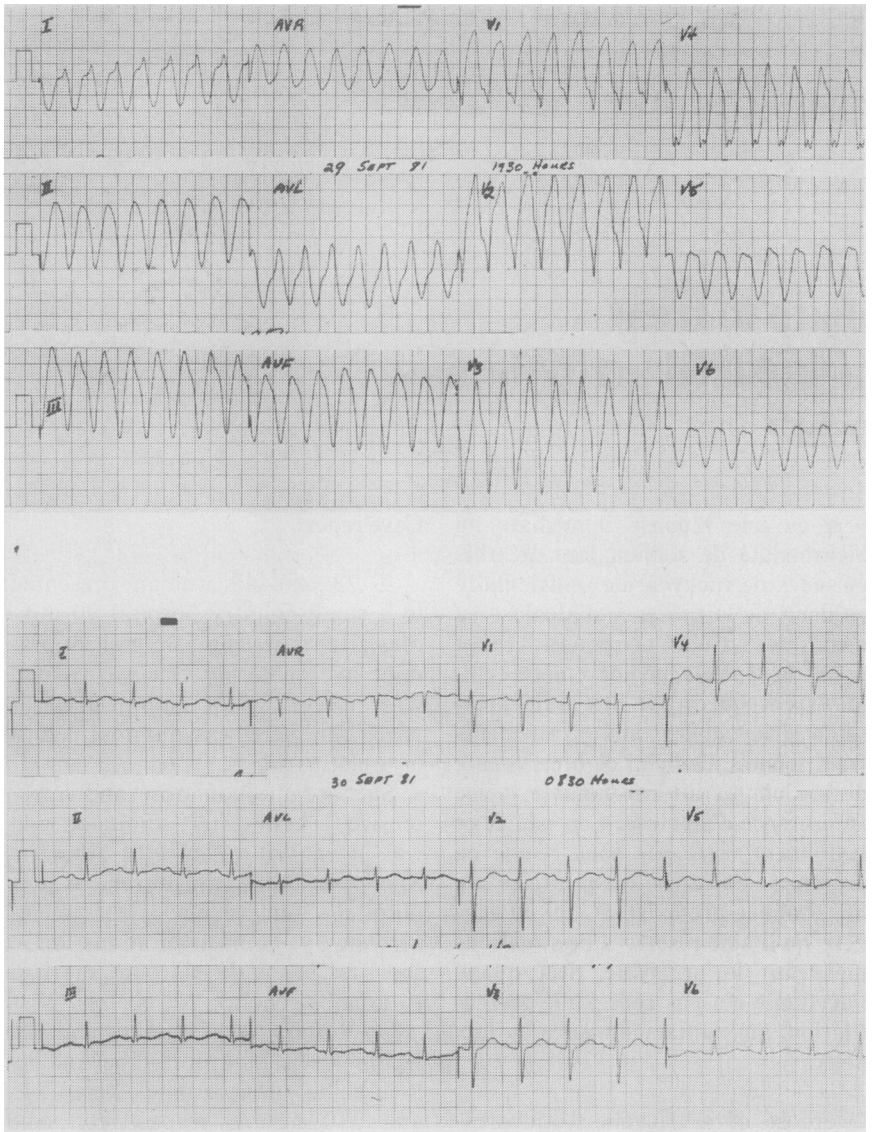


**Fig. 2—Normal ECG approximately 12 hours after treatment with sodium bicarbonate.**

physostigmine, but when 3 minutes had passed with no response another 50 mmol of sodium bicarbonate was administered and sinus rhythm returned.

While being transferred to the intensive care unit the patient had a major generalized seizure, which was successfully treated with an intravenous dose of 10 mg of diazepam. The pH on her arrival at the intensive care unit was 7.47; in spite of this, ventricular tachycardia/flutter recurred. Hyperventilation was continued, but sinus rhythm was not restored until a third dose of 50 mmol of sodium bicarbonate was given. The pH then rose to 7.66.

Further treatment included the administration of activated charcoal and magnesium sulfate, as well as positive pressure hyperventilation. There were no further arrhythmias, and the patient recovered completely and uneventfully. The following day the ECG was normal (Fig. 2).

Table I summarizes the clinical course and the blood gas data.

## Discussion

The patient described in this case report had consumed 5.35 g of imipramine hydrochloride and subsequently had life-threatening arrhythmias. Various forms of therapy have been advocated under these circumstances. Physostigmine has been used but carries the risk of increasing conduction blocks and

**Table I—Clinical and blood gas data for patient who had taken an overdose of imipramine hydrochloride**

| Time (pm) | Clinical status | Blood pressure (mm Hg) | pH | Partial pressure in arterial blood (mm Hg) | | Bicarbonate level (mmol/L) |
|---|---|---|---|---|---|---|
| | | | | Oxygen | Carbon dioxide | |
| 7:30 | Ventricular tachycardia on arrival in emergency department | 90/palpable | 7.36 | 289 | 30 | 17 |
| 7:42 | Sinus rhythm after first dose of sodium bicarbonate | 110/palpable | 7.52 | 376 | 22 | 18 |
| 7:50 | Ventricular tachycardia; no response to physostigmine, 2 mg | 80/palpable | – | – | – | – |
| 7:55 | Sinus rhythm after second dose of sodium bicarbonate | 80/50 | 7.58 | 247 | 25 | 24 |
| 8:20 | Ventricular tachycardia after transfer to intensive care unit | 90/palpable | 7.47 | 256 | 29 | 21.2 |
| 8:40 | Sinus rhythm after third dose of sodium bicarbonate | 90/50 | 7.66 | 241 | 22 | 24.7 |
| 9:25 | Sinus rhythm 45 minutes after last dose of sodium bicarbonate | 100/50 | 7.54 | 187 | 29 | 24.8 |

producing seizures.[5,6] Phenytoin and lidocaine have also been advocated, but they are not always effective and may have toxic effects, such as conduction disturbances.[5] Several authors have recommended the use of sodium bicarbonate as the first choice because of its safety and efficacy.[1]

We were presented with an unusual situation. The patient had metabolic acidosis, a normal anion gap (measured within 20 minutes of admission) and, in spite of superimposed metabolic and respiratory alkalosis, recurrent life-threatening arrhythmias. It was not possible to determine whether the acid–base changes that occurred during treatment resulted solely from the sodium bicarbonate therapy or were secondary to improved hemodynamics. What is important is that despite a pH greater than 7.35, the administration of sodium bicarbonate, with the expected increase in pH, was associated with reversion to sinus rhythm on three occasions.

Although hyperventilation alone has some theoretical advantages over sodium bicarbonate administration because of the difference in cell permeability between carbon dioxide and bicarbonate,[3] in the present case sodium bicarbonate appeared to have a rapid effect. Further hyperventilation, with a further decrease in the partial pressure of carbon dioxide and an increase in pH, might have produced the same outcome. This possibility was not addressed during the emergency treatment of our patient.

The mechanism by which the administration of sodium bicarbonate reverses life-threatening arrhythmias associated with overdose of a tricyclic antidepressant is unknown. Whether it is related to a change in pH, an increase in the osmolar load or an increase in the circulating amount of sodium[7] is speculative, but the known increase in protein binding, with a corresponding decrease in availability of the free drug, as the pH increases is a plausible mechanism.[3]

In the case we have described, ventricular tachycardia in a patient with an elevated pH was reversed immediately after sodium bicarbonate administration. This suggests that an elevated pH does not preclude effective treatment with sodium bicarbonate. The potential hazards of combined metabolic and respiratory alkalosis must be considered before this treatment is instituted in such a case, but the frequent lack of response of this type of arrhythmia to conventional antiarrhythmic therapy makes the use of sodium bicarbonate reasonable under these circumstances. This is particularly true when a life-threatening arrhythmia has failed to respond to conventional therapy.

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