METABOLISM OF SODIUM NITROPRUSSIDE AND CYANIDE IN THE DOG

C. J. VESEY, P. J. SIMPSON, L. ADAMS AND P. V. COLE

SUMMARY

Blood cyanide (HCN) and thiocyanate (SCN) concentrations were measured at intervals in anaesthetized dogs given bolus doses of sodium nitroprusside (SNP) 1 mg kg\(^{-1}\) or potassium cyanide 1.07 mg kg\(^{-1}\) and in animals infused with SNP 1.5 mg kg\(^{-1}\) for 1 h. Cyanide appeared rapidly in the red cells to give peak concentrations which accounted for more than 90% of the total blood HCN. A delay between the peak plasma and red cell HCN concentrations confirmed that some of the SNP was degraded in the plasma. Comparison of HCN and SCN concentrations with those measured previously in patients receiving an infusion of SNP suggests that the degradation of SNP and detoxication of HCN may be more rapid in the dog. The various pathways of HCN detoxication are discussed in relation to the reduced formation of SCN in dogs receiving SNP compared with those receiving KCN.

Since Hermann (1886) noted an intense odour of cyanide (HCN) in the body cavities of animals killed with sodium nitroprusside (SNP) some workers have believed that nitroprusside exerts its hypotensive effect by the production of minor degrees of cyanide poisoning. Johnson (1929) suggested that the hypotensive action was probably a result of the nitrosyl (NO) group of the SNP molecule and excluded cyanide as the cause of both the toxic and pharmacological effects of the drug. Subsequent work showed that cyanide was released from SNP on incubation with tissues (Hill, 1942) and more recently it has been demonstrated in man that infusion of the drug results in increased blood HCN concentrations (Vesey et al., 1974; Davies et al., 1975a; Vesey, Cole and Simpson, 1976; du Cailar, Mathieu-Daude and Deschodt, 1977; Smith et al., 1977). In addition a number of fatalities have been attributed to the release of HCN from infused SNP (Jack, 1974; Merrifield and Blundell, 1974; Davies et al., 1975; Vesey and Cole, 1975). As a consequence, maximum doses of SNP have been suggested (Editorial, 1975; Davies et al., 1975a; Vesey, Cole and Simpson, 1976; Bennett and Abbott, 1977; Michenfelder, 1977).

The metabolism and physiological effects of the drug have been studied in three groups of dogs with particular reference to cyanide and thiocyanate (SCN) production (Simpson et al., 1977; Simpson, 1978).

One group of dogs received bolus doses of SNP and a second group was given equivalent amounts of cyanide in the form of bolus doses of potassium cyanide (KCN). Plasma HCN and blood lactate concentrations and oxygen consumption were measured in a third group of dogs, infused with SNP 1.5 mg kg\(^{-1}\) at a constant rate for 1 h, to obtain an indication of the safety of the recommended maximum total dose for short term infusions.

This paper describes the changes in blood HCN and plasma SCN concentrations; a second paper reports on the associated cardiovascular and acid base changes (Simpson et al., 1979).

METHODS

Mongrel dogs were used in all three experimental groups. Details of the mean weights of the dogs and the mean dose of SNP or KCN are shown in table I. The lethal i.v. bolus dose of SNP for the dog is reported to be about 3.36 \(\mu\)mol kg\(^{-1}\) (1 mg kg\(^{-1}\)) (Johnson, 1929). This would release the same amount of HCN as would KCN 16.8 \(\mu\)mol kg\(^{-1}\) (1.09 mg kg\(^{-1}\)). Since this is well below the amount absorbed (40 \(\mu\)mol kg\(^{-1}\) or 2.6 mg kg\(^{-1}\)) from a lethal oral dose of KCN in the dog (Gettler and Baine, 1938), the animals in group 1 were each given an i.v. bolus dose of SNP 3.36 \(\mu\)mol kg\(^{-1}\) in a total volume of 2 ml 0.9% saline and those in group 2 received a mean bolus...
Table 1. Experimental groups

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of dogs</th>
<th>Weight (kg) Mean</th>
<th>Range</th>
<th>Agent administered</th>
<th>Dose (µmol kg⁻¹ ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>23.63</td>
<td>16.3-36.3</td>
<td>bolus SNP</td>
<td>3.36</td>
</tr>
<tr>
<td>2</td>
<td>6</td>
<td>21.94</td>
<td>16.8-25.9</td>
<td>bolus KCN</td>
<td>16.46 ± 0.46</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>20.65</td>
<td>15.2-33.6</td>
<td>SNP infused for 1 h</td>
<td>5.1 ± 0.23</td>
</tr>
</tbody>
</table>

dose of KCN 16.46 µmol (1.07 mg kg⁻¹) dissolved in 1–2 ml of isotonic saline. The concentrations of the freshly prepared KCN solutions were determined in duplicate before and after administration of an appropriate volume and the mean values used to calculate the exact dose given to each dog.

McDowall and colleagues (1974) found that SNP 5.37 µmol kg⁻¹ h⁻¹ (1.6 mg kg⁻¹ h⁻¹) was the highest dose associated with recovery in baboons, therefore the dogs of group 3 were infused for 1 h with a mean dose of SNP 5.1 µmol kg⁻¹ (1.52 mg kg⁻¹). The solution was freshly prepared and administered in 100 ml of isotonic saline from a foil-wrapped bottle by means of an IVAC 201 drip controller. A check was made on the exact volume administered on each occasion by weighing the bottle before and after the infusion.

Anaesthesia was induced with pentobarbitone sodium 25 mg kg⁻¹ and maintained with 66% nitrous oxide in oxygen. The animals were kept in a lateral position and the lungs ventilated to PaCO₂ 4.5–5.2 kPa and incremental doses of pentobarbitone (12.5 mg kg⁻¹; 2–4 doses per experiment) were administered as necessary. Pentobarbitone was not given immediately before making measurements, in view of its occasional transient hypotensive effects. Body temperature was monitored with a rectal thermometer and maintained constant by the use of a warming blanket.

A solution of 0.18% saline in 4% dextrose was infused into one foreleg vein to replace blood lost through sampling. The superficial femoral artery of one leg was cannulated for continuous recording of systemic arterial pressure and for sampling arterial blood. The common femoral artery was left intact to prevent the development of ischaemia and localized acidosis in the limb. A catheter was introduced to the femoral vein to ensure that drugs were given centrally. Central venous pressure was monitored via a catheter inserted to the external jugular vein.

A Swan–Ganz catheter was placed in the pulmonary artery of three dogs in each of groups 1 and 2, and in all dogs in group 3. E.g., systemic arterial, pulmonary arterial and central venous pressures were recorded continuously using a Devices M19, six-channel recorder.

When a stable state had been reached, simultaneous heparinized samples of mixed venous blood and arterial blood were obtained. Further samples were taken at the following times after the bolus injections (groups 1 and 2): 2, 5, 10, 20, 40, 80, 120, 160 and 240 min. Arterial and venous blood-gas measurements were made with an Instrumentation Laboratories 413 automatic blood-gas analyser. No therapeutic correction was made for the development of metabolic acidoses which occurred in some dogs and which recovered spontaneously in all but one.

In the third group of dogs the same experimental procedure was used. Physiological measurements and blood sampling were undertaken before infusion, at 15, 30, 45 and 60 min during infusion and at 10, 30, 60, 120 and 180 min after termination of the infusion.

The samples of arterial blood, collected in ice-cooled syringes, were centrifuged immediately for 10 min at 2300 rev min⁻¹ and 0 °C. The plasma and erythrocytes were separated and analysed for cyanide and thiocyanate by methods described previously (Vesey, Cole and Simpson, 1976).

RESULTS

There was a slower change in both plasma and red cell HCN concentrations and a much smaller plasma HCN concentration following boluses of SNP than following KCN (figs 1, 2). Similarly there was both a slower increase in plasma SCN and a lower peak SCN concentration in group 1 than in those animals receiving a bolus dose of KCN (fig. 3).

The method used for determining plasma SCN (Aldridge, 1945) also measures HCN. Plasma thiocyanate concentrations are much greater than plasma HCN concentrations and it is necessary to dilute the plasma for measurement of SCN. This dilution, in addition to the loss of plasma HCN during storage in deep freeze, results in an insignificant
contribution to the SCN result by the cyanide present. However, the dogs of group 2 had very large initial plasma HCN concentrations and the sharp peak at the start of the SCN curve (fig. 3A) is a result of some cyanide remaining after storage at −20 °C. The increase in SCN at this point was obtained by extrapolation and is shown as a dotted line.

The mean plasma and red cell cyanide concentrations and the increase in plasma SCN, measured at intervals during and for 3 h after infusion, are shown in figure 4. The larger amount of SNP given to group 3 dogs results in proportionately greater peak red cell HCN and plasma SCN concentrations than occurred in group 1. As expected, the slow infusion of the SNP dose resulted in a smaller plasma cyanide concentration, although the time scale of the increase in plasma SCN was similar to that of group 1. Both the plasma and red cell HCN concentrations reached a maximum at the end of infusion with a distinct decrease after 10 min.

The plasma HCN concentration curve appears to flatten out 15 min before the end of the infusion. The peak plasma HCN concentration occurred
**TABLE II. Comparison of maximum blood cyanide concentrations (μmol litre⁻¹, mean ± SEM) in dogs receiving either potassium cyanide (KCN) or sodium nitroprusside (SNP)**

<table>
<thead>
<tr>
<th></th>
<th>Bolus KCN*</th>
<th>Bolus SNP*</th>
<th>Infused SNP†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of measured peak plasma HCN (min)</td>
<td>2</td>
<td>5-10</td>
<td>45-60</td>
</tr>
<tr>
<td>Time of measured peak RBC HCN (min)</td>
<td>5</td>
<td>20</td>
<td>60 (end of infusion)</td>
</tr>
<tr>
<td>Peak plasma HCN</td>
<td>26.4 ± 6.4</td>
<td>2.2 ± 0.3</td>
<td>1.53 ± 0.16</td>
</tr>
<tr>
<td>Peak plasma HCN as % of total blood HCN</td>
<td>17.9</td>
<td>3.2</td>
<td>1.52</td>
</tr>
<tr>
<td>Peak RBC HCN</td>
<td>124 ± 11.5</td>
<td>95.3 ± 4.6</td>
<td>119.0 ± 8.4</td>
</tr>
<tr>
<td>Peak RBC HCN as % of total blood HCN</td>
<td>91.5</td>
<td>98.4</td>
<td>98.7</td>
</tr>
<tr>
<td>Plasma HCN at time of peak RBC HCN</td>
<td>11.5 ± 3.1</td>
<td>1.6 ± 0.35</td>
<td>1.54 ± 0.18</td>
</tr>
<tr>
<td>RBC HCN at time of peak plasma HCN</td>
<td>121.3 ± 6.7</td>
<td>67.0 ± 4.4</td>
<td>99.0 ± 7.0</td>
</tr>
</tbody>
</table>

* Following the bolus dose blood samples were taken at 2, 5, 10, 20, 40, 60, 120, 160 and 240 min.
† During infusion blood samples were taken at 15-min intervals and after infusion at 10, 30, 60, 120 and 180 min.

before the peak red cell cyanide and a higher proportion of the total blood HCN was present in the red cells following SNP (table II).

One of the dogs in group 2 died 52 min after a bolus dose of KCN 17.85 μmol kg⁻¹ (1.16 mg kg⁻¹). The peak plasma HCN was 43.5 μmol litre⁻¹ and the peak red cell cyanide was 123.2 μmol litre⁻¹ 2 min after the dose. Similar values were measured in one other dog which received KCN 16.77 μmol kg⁻¹ (1.09 mg kg⁻¹) and survived for the duration of the experiment.

**DISCUSSION**

The main details of the metabolic pathway of SNP were described by Page and colleagues (1955) and have been reviewed by Davies and others (1975a) and Tinker and Michenfelder (1976). Our understanding of the metabolic processes involved are shown in figure 5. Apart from doubt as to the existence of “thiocyanate oxidase” (Vesey and Wilson, 1978), an enzyme thought to be present in the red cells and responsible for the conversion of SCN to HCN (Goldstein and Reiders, 1953), several uncertainties remain.

As Page and others (1955) suggested, SNP probably reacts with sulphydryl groups (—SH), breaking down to release HCN. In contrast with their data, this reaction occurs to a significant degree in plasma in vitro, although it is more rapid in the presence of erythrocytes (Vesey, Cole and Simpson, 1977). Smith and Kruszyna (1974) have proposed that the intact nitroprusside ion (Fe(CN)₅NO⁻₂⁻) enters the red cell and reacts with haemoglobin to form one molecule of cyanmethaemoglobin and four molecules of HCN which can then diffuse into the plasma.

The fact that HCN accumulates rapidly in the red cell is illustrated by the results for group 2 (fig. 2, table II). The peak red cell cyanide concentration was five times that of the maximum measured plasma HCN concentrations and occurred within 5 min of injection of a bolus of KCN. In those animals receiving a bolus dose of SNP, the peak red cell HCN

![Figure 4](https://academic.oup.com/bja/article-abstract/51/2/89/240527/1520721027)
similarly occurred after that in the plasma and increased to a value more than 40 times that of the peak plasma HCN (fig. 1, table II). The occurrence of this peak red cell cyanide concentration some 10 min after that in the plasma and the higher proportion of blood cyanide in the red cells after nitroprusside would suggest that breakdown of SNP occurs in both the plasma and the erythrocytes. It is suggested that 20–30% of the breakdown of SNP occurs in the plasma (fig. 5). However, the values for the relative contributions made by the red cells and plasma to the decomposition of SNP in blood are based on in vitro studies and it is uncertain if they apply in vivo (Vesey, Cole and Simpson, 1977).

In patients 98.4% of the total blood cyanide was found in the red cells at the end of infusion (Vesey, Cole and Simpson, 1976). Similar figures were obtained for dogs given SNP (group 1: 98.4% when r.b.c. HCN reached a peak, and group 3: 98.7% at the end of infusion).

After 60 min infusion of SNP (group 3) the increase in mean plasma and red cell HCN concentration above baseline values were $1.36 \pm 0.18$ and $118.4 \pm 8.3 \mu$mol litre$^{-1}$ ($\pm$ SEM) respectively. Our previous work (Vesey, Cole and Simpson, 1976) indicates that, following infusion of SNP in man, such values would be expected after total doses of SNP 2.2 and 3.13 $\mu$mol kg$^{-1}$ respectively (0.66 and 0.93 mg kg$^{-1}$), whereas the dogs received an infused dose of SNP 5.1 $\mu$mol kg$^{-1}$ (1.52 mg kg$^{-1}$). This would suggest either that SNP breakdown occurs at a slower rate in the dog or that both SNP decomposition and cyanide metabolism are more rapid in the dog than in man. When the plasma and red cell HCN concentrations 1 h after infusion are compared with those for patients (table III) the mean values in the dogs are smaller, although the mean infused dose of SNP was greater and the period of infusion shorter in the dogs. The decrease in plasma HCN concentration to less than preinfusion values after only 3 h emphasizes the rapid detoxication of HCN in the dog.

Aitken and colleagues (1977) reported that the mean blood cyanide concentrations for 13 patients receiving an infusion of SNP reached a peak within 45 min after the end of infusion. This contrasts with our findings in the dogs, in which the HCN concentration had decreased by 10% 10 min after infusion. These differences may result from the slower metabolism of SNP and HCN in man. However, this may not be the full explanation, since although the highest mean blood cyanide concentration at the end of infusion in our 26 patients (Vesey, Cole and Simpson, 1976) was similar to that measured by Aitken and colleagues (1977), (HCN 25 $\mu$mol litre$^{-1}$ blood and
24 \mu mol \text{ litre}^{-1} \text{ respectively}, 1 \text{ h} \text{ after the end of infusion the mean concentrations had decreased by 50\% in our study compared with a 20\% decrease in their patients. The differences may be a result of the dissimilar mean infusion times (86 min and 35.5 min respectively), although the mean doses of SNP administered were also quite different (0.42 mg \text{ kg}^{-1} \text{ compared with 0.22 mg \text{ kg}^{-1} \text{ respectively}). A further complicating feature is that, whereas Aitken measured whole blood cyanide in frozen samples, we determined plasma and red cell cyanide separately in freshly taken whole blood to avoid artefactual formation of HCN from SCN (Vesey and Wilson, 1978), which is probably enhanced by freezing and thawing of \textit{whole blood} (Ballantyne, 1977).

There are at least six known ways by which HCN may be removed from the blood (fig. 5). The major part is converted to thiocyanate (table III). It is considered generally that this is brought about by the mitochondrial enzyme, rhodanase (Sorbo, 1962). The observation that, 3 h after infusion, the plasma HCN concentrations were smaller than the preinfusion values in all the dogs (table III) might suggest that the enzymes involved in detoxification may be induced by exposure to HCN. However, rhodanase activity is not altered following chronic exposure to cyanide in man (Osuntokun, 1972) or in rats (C. Vesey and J. Wilson, unpublished observations).

Comparison of the increase in plasma SCN concentrations in the dogs with those reported previously for patients (Vesey, Cole and Simpson, 1976) suggests also that cyanide detoxication is more rapid in the dog. Thiocyanate concentrations measured in patients 1 h after infusion showed a significant correlation with the total dose of SNP \((r = 0.68, \ P < 0.001, \ y = 1.6 + 6.5x \ (\mu mol \text{ litre}^{-1}))\). A total dose of SNP 5.1 \mu mol \text{ kg}^{-1} (1.5 mg \text{ kg}^{-1}) should produce, therefore, an increase in SCN of 35 \mu mol \text{ litre}^{-1} plasma. In the dogs, however, plasma concentrations 1 h after infusion had increased by 45.6 \pm 4.3 \mu mol \text{ litre}^{-1} (mean \pm \text{ SEM}) in spite of the fact that the dog has a larger thiocyanate space than man (Altmann and Dittmer, 1971).

The increase in plasma SCN in the dogs infused with SNP (group 3) was much greater than the increase in blood concentration reported by Michenfelder (1977) in a similar study (SCN 50 \mu mol \text{ litre}^{-1} \text{ plasma compared with about 7 \mu mol \text{ litre}^{-1} \text{ blood} 2 \text{ h} \text{ after the end of infusion}). Thiocyanate concentrations would be expected to be much greater than 7 \mu mol \text{ litre}^{-1} since the dogs in Michenfelder's study received 50\% more SNP (a total dose of 167.8 \mu mol SNP (50 mg) compared with a mean of 105.7 \pm 12.8 \mu mol (31.5 mg) in our dogs). In addition, Michenfelder found that there was no difference in blood SCN concentrations between dogs with 10\% of their
haemoglobin as methaemoglobin and untreated animals following infusion with SNP. In the former group, 60% of HCN was trapped as cyanmethaemoglobin, which would be expected to result in a concentration of SCN smaller than in the untreated dogs. In fact Smith, Mukerji and Seabury (1940) showed that SCN formation was delayed for 2 h in dogs given a dose of sodium nitrite before an injection of sodium cyanide. These discrepancies may be accounted for by the fact that Michenfelder determined blood rather than plasma SCN. When whole blood is deproteinized with trichloroacetic acid, 80–90% of the SCN is lost; a small amount is converted to HCN (Vesey and Wilson, 1978), whilst the majority is carried down with the precipitated haemoglobin (Goldstein, 1950).

The major part of red cell cyanide is converted to SCN (table IV). This is confirmed by the close correlation between the decrease in mean red cell HCN and the increase in mean plasma SCN in all three groups of dogs (fig. 6). The question remains as to how the red cell cyanide is converted to thiocyanate. The enzyme β-mercaptopyruvate sulphur transferase, which converts HCN to SCN, occurs in red cells (Van den Hamer, Morell and Scheinberg, 1967). However, it seems unlikely that this enzyme contributes significantly to the formation of thiocyanate at this stage, since supply of the substrate β-mercaptopyruvate will be a limiting factor in the erythrocyte. In addition, the red cell seems to be relatively impermeable to SCN (Vesey and Wilson, 1978). It is probable, therefore, that the red cell HCN passes out unchanged into the plasma, and thence into the tissues to be converted to SCN.

The proportion of the cyanide dose converted to SCN was smaller in both groups of dogs receiving SNP than in the group given KCN (table IV). Small amounts of HCN are excreted in expired air (Boxer and Rickards, 1952a; Vesey, Cole and Simpson, 1976) and urine (Boxer and Rickards, 1952b). In addition, small amounts of cyanocobalamin (CNB₁₂) are formed (Vesey et al., 1974), some cyanide from which is channelled into 1-carbon pathways whilst the rest of the CNB₁₂ is excreted in the urine (Boxer and Rickards, 1952b). It has been postulated also that SCN is formed from CNB₁₂ to release hydroxocobalamin (Wokes and Ellis, 1966) which is then recycled, but the evidence is inconclusive. Contrary to some reports (Tinker and Michenfelder, 1976), rhodanase is not dependent on B12 for its action. The conversion of some HCN to cyanate (CNO) and thence to carbon dioxide is a further possibility (Williams, 1959). These additional mechanisms for the removal of HCN would account for only small amounts of cyanide, perhaps some of the 20% not accounted for as SCN in the dogs receiving KCN, but there is no apparent reason why there should be an increased metabolism by these routes following SNP.

The HCN could be trapped as cyanmethaemoglobin following a dose of SNP, as Smith and Kruszyna (1974) suggest. This possibility is not evident from our results since less than 3% of the cyanide dose in all three groups of dogs remained in the red cells when plasma SCN concentrations reached a maximum (table IV).

The metabolically inert compound 2-iminothiazolidine-4-carboxylic acid (ITC) has been detected in the urine of man and of rats exposed to cyanide

<table>
<thead>
<tr>
<th>Group</th>
<th>Time between dose and peak plasma SCN (min)</th>
<th>% of dose as SCN*</th>
<th>% of dose as RBC HCN*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1—SNP bolus</td>
<td>160</td>
<td>63</td>
<td>2.6</td>
</tr>
<tr>
<td>2—KCN bolus</td>
<td>120</td>
<td>82</td>
<td>0.6</td>
</tr>
<tr>
<td>3—SNP infusion</td>
<td>120†</td>
<td>64.5</td>
<td>1.8</td>
</tr>
</tbody>
</table>

* Calculated from the concentrations measured at the time of maximum plasma SCN.
† Time from the end of infusion.
(Wood and Cooley, 1956). This compound arises by reaction between HCN and cystine (a disulphide, \(-\text{S-S}\)-), and cysteine (\(-\text{SH}\)) is a by-product of the process (fig. 5). Since some SNP is thought to break down by reaction with \(-\text{SH}\) groups, presumably with the formation of \(-\text{S-S}\)-, it is possible that more HCN will be converted to ITC (possibly encouraged by a cyclic process involving \(\frac{\text{SNP}}{\text{HCN}}\))

cystine\(-\text{cysteine}\) than after KCN.

Our results would suggest that at least some of the breakdown of SNP occurs in the plasma and that both the breakdown of SNP and the subsequent detoxication of the cyanide are more rapid in the dog than in patients and thus, although the dog is a useful model for studying the metabolism of SNP and cyanide, caution is required in the extrapolation of these results to patients.

In addition, the major proportion of the blood cyanide arising from SNP is found in the red cells and most of it is converted to SCN; however, a significant proportion of the cyanide from a dose of SNP in dogs is not detoxicated to SCN and this could result from enhanced formation of ITC.

REFERENCES


---


---


---


---

METABOLISM OF NITROPRUSSIDE

---

METABOLISME DU NITROPRUSSIATE DE SODIUM ET DU CYANURE CHEZ LE CHIEN

---

RESUME

On a mesuré à intervalles les concentrations de cyanures (HCN) et de thiocyanate (SCN) dans le sang de chiens anesthésiés auxquels on avait administré des doses de nitroprussiaste de sodium à raison de 1 mg kg⁻¹ ou de cyanure de potassium à raison de 1,07 mg kg⁻¹, ainsi que sur des animaux auxquels on avait infusé du SNP pendant 1 h à raison de 1,5 mg kg⁻¹. Le cyanure est apparu rapidement dans les globules rouges pour former des concentrations de pointe qui ont représenté plus de 90% du HCN contenu dans le sang. Le retard constaté entre les concentrations de HCN dans les globules rouges et dans le plasma confirme qu’une certaine quantité de SNP se dégrade dans le plasma. La comparaison des concentrations de HCN et de SCN avec celles mesurées antérieurement chez les malades recevant une infusion de SNP laisse penser que la dégradation du SNP et la désintoxication du HCN peut être plus rapide chez le chien. Les divers modèles de désintoxication du HCN sont décrits dans cet article en relation avec la formation diminuée de SCN chez les chiens recevant du SNP, par rapport à ceux recevant du KCN.

---

METABOLISMO DE NITROPRUSIATO SODICO Y CIANURO EN EL PERRO

---

SUMARIO

Se midieron a intervalos las concentraciones de cianuro (HCN) y tiocianato (SCN) en la sangre de perros anestesiados que recibieron dosis en bolo de nitroprusiato sódico (SNP) 1 mg kg⁻¹ o cianuro potásico 1,07 mg kg⁻¹ y de animales infundidos con SNP 1,5 mg kg⁻¹ durante 1 h. El cianuro apareció rápidamente en las células rojas hasta alcanzar concentraciones máximas que correspondieron a más de un 90% del total de HCN en la sangre. La plasma se demoró más que las células rojas en alcanzar la concentración máxima de HCN, lo cual confirma que parte del SNP se degradó en la plasma. Una comparación entre las concentraciones de HCN y SCN y aquellas medidas anteriormente en pacientes que recibían una infusión de SNP sugiere que la degradación de SNP y la destoxicación de HCN pueden producirse con mayor rapidez en el perro. Se discuten las diversas vías de destoxicación de HCN en relación a la reducida formación de SCN en los perros que recibían SNP a diferencia de aquellos que recibían KCN.