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CENTRAL ACTIONS OF BLOOD-BORNE ANGIOTENSIN II

by

MAW-CHANG LEE

DISSERTATION

Submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

PHYSIOLOGY

in the

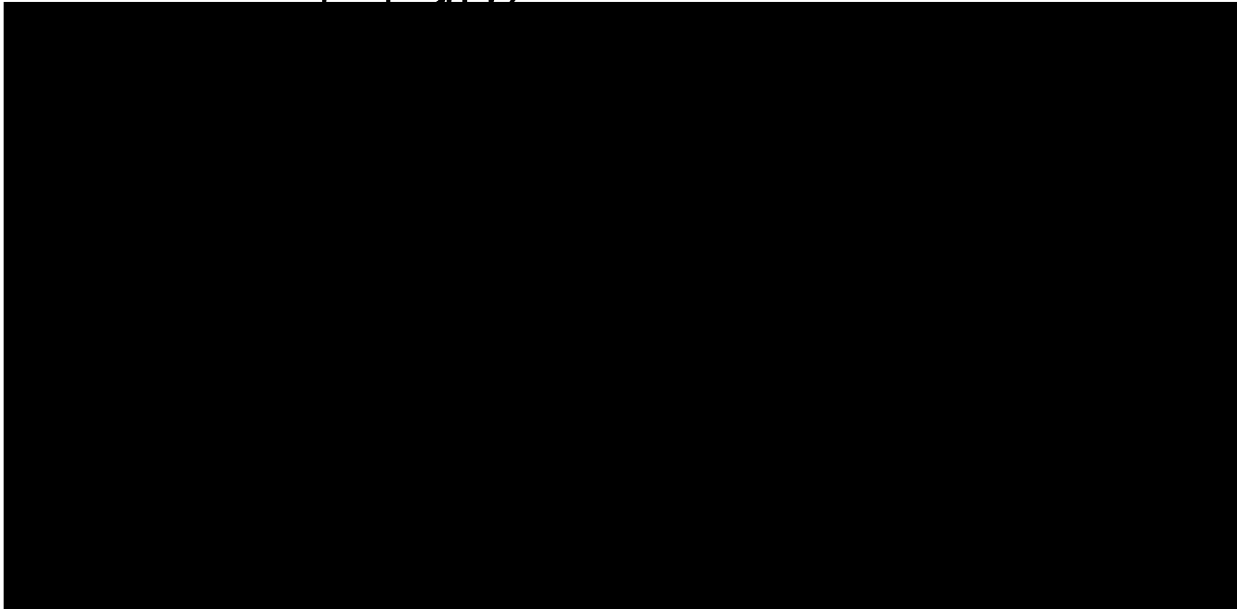
GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA

San Francisco

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CENTRAL ACTIONS OF BLOOD-BORNE ANGIOTENSIN II

MAW-CHANG LEE

ABSTRACT

Centrally-mediated pressor and drinking responses to blood-borne angiotensin II (AII) are described in this dissertation. In chronically-catheterized conscious mongrel dogs, infusion of AII into both common carotid arteries at 1.0 and 2.0 ng/kg/min for 10 min caused a greater increase in arterial blood pressure than i.v. AII. Intracarotid AII at the higher dose reduced common carotid blood flow to half, presumably due to its vasoconstrictor action on the extracranial vascular beds. According to a theoretical analysis, this local vasoconstriction could lead to a pressor response large enough to account for that to intracarotid AII.

The mean blood flow in each internal carotid artery in conscious dogs determined by the radioactive microsphere method was 7.7 ml/min, and that in the

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The first part of the text discusses the importance of maintaining accurate records of all transactions. It emphasizes that proper bookkeeping is essential for the success of any business, as it allows the owner to track income and expenses, and to identify areas where costs can be reduced. The text also mentions the need to keep records for tax purposes, as the IRS requires businesses to maintain detailed records of their financial activities.

The second part of the text focuses on the importance of staying organized and up-to-date on industry trends. It suggests that business owners should regularly read industry publications and attend trade shows to stay informed about new products, services, and market conditions. The text also highlights the importance of networking with other business owners in the industry, as this can lead to valuable opportunities for collaboration and growth.

The third part of the text discusses the importance of maintaining a strong customer base. It emphasizes that excellent customer service is a key factor in building a successful business, as satisfied customers are more likely to return and refer others to the business. The text also mentions the importance of keeping track of customer feedback and using it to improve products and services.

The fourth part of the text focuses on the importance of financial planning and budgeting. It suggests that business owners should create a budget for each year and stick to it as closely as possible. The text also mentions the importance of regularly reviewing financial statements and adjusting the budget as needed to ensure the business remains profitable.

The fifth part of the text discusses the importance of staying motivated and focused on long-term goals. It emphasizes that business ownership can be challenging, and it is important to stay committed and persistent in the face of adversity. The text also mentions the importance of seeking support from family, friends, and professional advisors when needed.

.....

The text concludes with a final thought on the importance of continuous learning and growth. It emphasizes that the business world is constantly changing, and it is essential for business owners to stay current in their knowledge and skills. The text also mentions the importance of being open to new ideas and taking calculated risks to achieve long-term success.

anastomotic artery between the external and internal carotid arteries was 3.3 ml/min. Since evidence exists that blood in both arteries mixes well before reaching the cerebral microcirculation, the brain would receive significantly lower concentration of AII when AII was infused into the external carotid arteries than when infused into the common carotid arteries. This was supported by the findings on plasma vasopressin response to AII. However, arterial pressure and plasma corticosteroid concentration, used as an index of ACTH secretion, increased to the same degree following both routes of AII. Therefore, even though some of the evidence suggests that the pressor effect of intracarotid AII is due to extracranial vasoconstriction, definite conclusions cannot be made.

Intraventricular saralasin, a competitive antagonist of AII, inhibited the pressor response to intravertebral, but not to intracarotid and i.v., AII. This result provides direct evidence that the pressor effect of intracarotid AII is mediated by different mechanism than that of intravertebral AII. It also suggests that the central pressor action of intravertebral AII is not essential in the pressor effect of circulating AII.

In Sprague-Dawley rats, intraventricular saralasin specifically inhibited drinking to AII i.v.; however,

- Wiederholungsfragen sind Fragen, die in der Vorlesung oder in den Vorlesungsmaterialien bereits behandelt wurden. Diese Fragen sind oft einfacher zu beantworten, da sie sich auf das Gelernte beziehen.

- Neuere Fragen sind Fragen, die in der Vorlesung oder in den Vorlesungsmaterialien nicht behandelt wurden. Diese Fragen sind oft schwieriger zu beantworten, da sie sich auf neuere Erkenntnisse oder auf die Anwendung des Gelernten beziehen.

- Wichtigere Fragen sind Fragen, die in der Vorlesung oder in den Vorlesungsmaterialien als wichtiger hervorgehoben wurden. Diese Fragen sind oft schwieriger zu beantworten, da sie sich auf die Kernkonzepte des Faches beziehen.

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it had no significant effect on drinking following 24- and 30-h water deprivation and abdominal vena caval ligation. Therefore, circulating AII appears not to play an essential role in drinking following these two stimuli.

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CHAPTER I GENERAL INTRODUCTION

Renin was first named by Tigerstedt & Bergman (1898) to indicate a pressor substance found in saline extracts of rabbit kidney. Its physiological significance was not appreciated until after the pioneering studies of Cash (1926) and Goldblatt et al. (1934). Cash suggested that a pressor substance from the kidney played a role in the pathogenesis of experimental hypertension following bilateral ligation of the renal arteries in dogs. Goldblatt and his colleagues successfully developed persistent hypertension by constricting the main renal arteries in dogs. He subsequently showed that this type of hypertension is due primarily to a humoral mechanism initiated by the ischemia of the kidney (Goldblatt 1937). This important work of Goldblatt led immediately to the search for the pressor substance. It soon led to the discovery that renin is an enzyme and that the pressor action of renin is due to the formation of angiotensin, the true pressor substance, almost simultaneously by Page & Helmer (1940) and Braun-Menendez et al. (1940a). Angiotensin was later found to exist in two different forms, angiotensin I and II (Skeggs et al. 1954a). They were then purified (Skeggs et al. 1954b, Bumpus &

THE PROBLEM OF THE FUTURE

The first thing I should mention is that I have been thinking about the future a lot lately. It's not just about the future in general, but about the future of our world and our lives. I feel like there's a lot of uncertainty out there, and I want to share some of my thoughts on what might happen and how we can prepare for it.

One of the biggest concerns I have is about the environment. Climate change is a real and present danger, and it's going to have a massive impact on our planet. We need to take action now to reduce our carbon footprint and transition to renewable energy sources. If we don't, the consequences will be catastrophic.

Another major concern is about technology. While it's brought us incredible progress, it's also creating new challenges. Privacy is being eroded, jobs are being lost to automation, and there's a growing digital divide. We need to ensure that technology is used for the benefit of all, not just a select few.

I also think about the future of our societies. There's a lot of political and social unrest around the world, and it's hard to see a bright future ahead. We need to focus on building a more just and equitable society, where everyone has a fair chance to succeed. Education is key here, as it's the best way to empower people and create a more resilient future.

On a personal level, I think about the future of my own life. I want to live a meaningful and fulfilling life, and I want to leave a positive legacy behind me. I need to stay focused on my goals, stay resilient in the face of adversity, and surround myself with good people. The future is uncertain, but I believe that with hard work and determination, I can make the most of whatever comes my way.

In conclusion, the future is both scary and exciting. It's a time of great challenges, but also of great opportunities. We need to work together to address the big issues facing our world and to create a better future for ourselves and for generations to come. Let's take action today, because the future is not something that just happens to us—it's something that we can shape.

Page 1954), their structure determined (Elliott & Peart 1957), and synthesized (Rittel et al. 1957, Schwarz et al. 1957).

It is now known that renin is a very specific carboxyl protease (Skeggs et al. 1977). Although renin-like activity has been identified in many organs (Peach 1977), the enzyme studied most intensively and of most physiological significance is secreted into the bloodstream by the kidney. Renal renin is localized in the juxtaglomerular cells of the kidney (Cook 1971). These granular cells are located in the media of the afferent arterioles as they enter the glomeruli. Renin secretion is stimulated following systemic hypotension, increased activity of the renal sympathetic nerves and following a reduction in the sodium load to the distal tubule of the kidney (Davis & Freeman 1976).

After being secreted into the bloodstream, renin acts on an α -globulin, called renin substrate or angiotensinogen, to form angiotensin I. Most evidence indicates that the angiotensinogen in the circulation is produced by the liver (Reid et al. 1978). The plasma concentration of angiotensinogen is increased by administration of adrenocortical steroids, estrogens and angiotensin II (AII), and by nephrectomy, ureteral ligation, hemodilution and hypoxia (Reid et al. 1978).

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Angiotensin I, a decapeptide, is converted to (AII), an octapeptide, after cleavage of the C-terminal dipeptide by converting enzyme. Although converting enzyme is ubiquitous (Erdős 1977), it has a very high activity in the lungs (Ng & Vane 1967). Most of the conversion from angiotensin I to II seems to occur during the passage of the blood through the lungs. Angiotensin II has a very short half-life in the blood, shorter than 1 min (Al-Merani et al. 1978). The enzymes that metabolize AII are generally termed angiotensinases. These include amino-, carboxy-, and endo-peptidases (Peach 1977). Angiotensinase activity is found in red blood cells and many tissues. One heptapeptide metabolite, [des-Asp¹] angiotensin II, is named angiotensin III and is biologically active.

There are various inhibitors of the renin-angiotensin system. Inhibitors of prostaglandin synthesis such as indomethacin and β -adrenergic blockers such as propranolol reduce renin secretion. The peptide pepstatin prevents renin from generating angiotensin I. Converting enzyme inhibitors such as SQ 20881 and SQ 14225 (catopril) inhibit conversion of angiotensin I to AII; however, they also inhibit the inactivation of bradykinin, a potent vasodilator. Analogs of AII such as saralasin are competitive inhibitors of AII

The first part of the report is devoted to a description of the
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 three main sections: (a) a general survey of the work done, (b) a
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at the receptor level (Ganong 1981, Laragh et al. 1977).

Although angiotensin I appears to be as potent as AII in stimulating catecholamine secretion from the adrenal medulla (Peach 1977) and angiotensin III no less potent than AII in stimulating aldosterone secretion from the adrenal cortex (Freeman et al. 1977), the exact physiological roles of angiotensins I and III are not established.

On the other hand, angiotensin II has many well-established actions, peripherally and centrally. In the periphery, blood-borne AII is a potent stimulator of aldosterone secretion (Davis 1974). In cross-circulation experiments in dogs, Davis (1963) and his coworkers discovered that a humoral factor is responsible for the release of aldosterone from the adrenal cortex. This humoral substance was subsequently shown to be AII by Laragh et al. (1960) and Biron et al. (1961). Blood-borne AII has direct constrictor action on vascular smooth muscles; it also increases sympathetic activity by acting on the adrenal medulla to stimulate catecholamine secretion (Braun-Menendez et al. 1940b, Feldberg & Lewis 1964), on the sympathetic ganglia to facilitate ganglionic transmission (Lewis & Reit 1965), and on the post-ganglionic nerve terminals

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to stimulate adrenergic transmitter release and to increase the responsiveness of smooth muscle to the transmitter (Zimmerman 1978). However, the vasoconstrictor effect of AII seems primarily to be due to its direct action on the vascular smooth muscle (Bohr 1974). Although AII has been called the most potent naturally occurring pressor agent, the maximum contraction produced by AII is generally less than that produced by α -adrenergic agonists (Bohr 1974). Angiotensin II constricts both pre- and post- capillary resistance vessels (Jarhult 1971), and its constrictor effect varies with different vascular beds.

Centrally, blood-borne AII stimulates secretion of vasopressin at least in some laboratories (Bonjour & Malvin 1970, Ramsay et al. 1978, Brooks et al. 1980) and ACTH (Maran & Yates 1977, Ramsay et al. 1978, Brooks et al. 1980). However, the physiological significance of these actions of AII is not established. Circulating AII also causes drinking and a centrally-mediated pressor response, which will be discussed in detail later. Angiotensin II administered into the lateral and third cerebroventricles also induces drinking and pressor response, and stimulates secretion of vasopressin (Severs et al. 1970, Mouw et al. 1971) and ACTH (Maran & Yates 1977). The sites of action of AII

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for increased secretion of vasopressin and ACTH have not been established. However, for vasopressin, the supraoptic nucleus (Nicoll and Barker 1971), the posterior pituitary (Gagnon et al. 1973) and the subfornical organ (Simpson 1981) have been suggested. For ACTH, the median eminence (Gann 1969) and the anterior pituitary (Maran & Yates 1977) have been implicated.

Circulating AII does not gain access to the cerebroventricles (Ramsay & Reid 1975, van Houten et al. 1980), neither does intraventricular AII gain access to the blood (Brightman et al. 1970). Therefore, the sites of action of intraventricular AII are likely to be different from those of circulating AII. Although the brain renin-angiotensin system has been postulated (Ganten et al. 1971, Fischer-Ferraro et al. 1971), its physiological significance and even its existence remain debatable. Therefore, the actions of blood-borne rather than intraventricular AII are studied in this dissertation.

The central pressor action of circulating AII was first demonstrated by Bickerton & Buckley (1961) in cross-circulation experiments in dogs. Its physiological significance was not appreciated until Yu & Dickson (1965) showed that AII infused into the vertebral arteries of conscious rabbits increased blood pressure

in doses that were ineffective when given intravenously. This finding was later confirmed in greyhounds (Lowe & Scroop 1969), man (Ueda et al. 1969) and mongrel dogs (Ferrario et al. 1970). This pressor response is rapid in onset and usually accompanied by an increase in heart rate. It is mediated mainly by increased efferent sympathetic activity in mongrel dogs (Ferrario et al. 1972) and rabbits (Yu & Dickinson 1971), and by decreased vagal tone to the heart in greyhounds (Scroop & Lowe 1969).

By transecting the brainstem at different levels, the site responsible for the pressor action of intravertebral AII was shown to lie in the medulla in dogs (Joy & Lowe 1970a). Since AII is unlikely to cross the normal blood-brain barrier (Brightman et al. 1970, Ramsay & Reid 1975, van Houten et al. 1980), in order to exert a central action, circulating AII must act on some brain locus which is outside the blood-brain barrier. Circumventricular organs, located around the ventricular system, are different from typical brain tissue in that they are highly vascularized with fenestrated capillaries (Weindl 1973). Ablation of the area postrema, a circumventricular organ in the caudal medulla, in dogs abolished the pressor response to intravertebral AII (Joy & Lowe 1970b, Gildenberg et

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al. 1973), reduced the pressor response to AII i.v. to half (Scroop et al. 1971), and impaired the ability to maintain blood pressure following hemorrhage (Katic et al. 1971) and the development of renal hypertension (Scroop et al. 1975). The area postrema was shown to be the most responsive site in the pressor effect to microinjection of AII in cats (Ueda et al. 1972). Electrical stimulation of the area postrema in dogs increased blood pressure and heart rate (Barnes et al. 1979). Therefore, the area postrema is generally taken as the site of action of the central pressor effect of circulating AII in dogs.

However, there is evidence that more rostral sites may also be involved in the central pressor action of AII. Intraventricular AII was shown to act on the subnucleus medialis in the midbrain to raise blood pressure in cats (Severs et al. 1967, Deuben & Buckley 1970). Transection of the midbrain abolished the pressor response to intraventricular, but not intravertebral, AII in dogs (Gildenberg et al. 1973). Microinjection of AII into the hypothalamus was reported to increase blood pressure in cats (Dutta et al. 1971). More recently, local injection of AII into the subfornical organ, a circumventricular organ adjacent to the dorsal third ventricle, has been shown to cause a pres-

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- Mr. Elizabeth Green, 1111 Madison Avenue, New York, N.Y.
- Mr. William Black, 1212 West End Avenue, New York, N.Y.
- Mr. Margaret Gray, 1313 East 86th Street, New York, N.Y.
- Mr. Thomas King, 1414 Central Park West, New York, N.Y.
- Mr. Patricia Lee, 1515 Riverside Drive, New York, N.Y.
- Mr. Daniel Hall, 1616 York Avenue, New York, N.Y.
- Mr. Susan Scott, 1717 Lexington Avenue, New York, N.Y.
- Mr. Christopher Young, 1818 Columbus Avenue, New York, N.Y.
- Mr. Rebecca Adams, 1919 Riverside Park, New York, N.Y.
- Mr. Steven Baker, 2020 Riverside Drive, New York, N.Y.
- Mr. Jennifer Wilson, 2121 York Avenue, New York, N.Y.
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- Mr. Ava Taylor, 3333 Columbus Avenue, New York, N.Y.
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pressor response in rats (Mangiapane & Simpson 1980). Angiotensin II infused into the carotid artery, rather than the vertebral artery, has been reported to elicit a greater increase in blood pressure than intra-aortic AII in rats (Haywood et al. 1980). This pressor effect was abolished by lesion of the anteroventral third ventricle (Fink et al. 1980), but not the area postrema (Haywood et al. 1980). The pressor response to AII i.v. was attenuated by subfornical organ lesion (Mangiapane & Simpson 1980), but not by area postrema lesion in rats (Zandberg et al. 1977, Buggy et al. 1977). Therefore, the area postrema does not seem to play an essential role in the central pressor action of circulating AII in rats.

Although intracarotid AII was generally reported to have no central pressor action in dogs (Severs & Daniels-Severs 1973), most of the studies were done on anesthetized preparations. Fitzsimons et al. (1978) reported that intracarotid AII increased blood pressure in 2 conscious dogs at a dose which was ineffective when given i.v. in 1 dog. However, they concluded, unjustifiably, that intracarotid AII induced no significant pressor response, despite of the small sample size. On the other hand, Reid (1980) recently showed that in conscious dogs intracarotid AII induced a pres-

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pressor response at doses that were ineffective when given intravenously. This pressor response differed from that produced by intravertebral AII in that it was smaller in magnitude, slower in onset, and not accompanied by an increase in heart rate. The mechanism of this pressor response is the main topic of the studies to be presented in Chapter III to V in this dissertation.

As renal renin is secreted into the bloodstream where AII is subsequently generated, blood-borne AII can increase blood pressure by acting on the systemic vascular beds directly, and by acting on the brain to exert a central pressor action. It is of considerable interest to determine the contribution of each component to the overall pressor effect of circulating AII. This question is examined in Chapter VI.

Another action of AII to be studied is its remarkable capacity to stimulate drinking. Renal renin was first suggested by Fitzsimons (1964) to play a role in thirst based on the finding that ligation of the abdominal vena cava was a less effective stimulus to drinking in nephrectomized rats than in normal ones. It was later found that intraperitoneal injection of saline extracts of renal cortex caused rats to drink and that the dipsogenic factor was inseparable from renin

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(Fitzsimons 1969). As saralasin inhibits drinking induced by renin and angiotensin I, the true dipsogenic component was later shown to be AII (Fitzsimons, Epstein & Johnson 1978). Intracranial AII is the most potent dipsogen ever studied (Booth 1968, Epstein et al. 1970, Ramsay & Reid 1975, Fitzsimons & Kucharczyk 1978). Although systemic administration of AII is less potent than intracranial AII in eliciting drinking, blood-borne AII at concentrations within the physiological range has been reported to cause drinking in rats and dogs (Hsiao et al. 1977, Fitzsimons et al. 1978).

The subfornical organ has been suggested to contain receptors which mediate drinking produced by circulating AII in rats (Simpson et al. 1978). The evidence includes the observation that the subfornical organ is one of the most sensitive sites in the brain in eliciting drinking to local injection of AII, that ablation of the organ eliminated drinking elicited by AII i.v., and that infusion of saralasin directly into the organ selectively antagonized drinking induced by AII i.v. In addition, iontophoretic application of AII to individual neurons of the organ increased their firing rates (Felix & Akert 1974), and this effect could be antagonized by saralasin (Phillips & Felix 1976). However, the organum vasculosum of the lamina ter-

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minalis, a circumventricular organ in the anteroventral third ventricle, has also been implicated as one of the receptor sites for the dipsogenic action of AII. Small amounts of AII applied to the region caused drinking in rats, and destruction of the region abolished drinking induced by intravenous or intraventricular AII (Phillips 1978). In addition, the preoptic area has been proposed to contain AII dipsogenic receptors (Mogenson et al. 1975). Further research is needed to determine the relative importance of these areas in the control of water intake.

Although studies on drinking following caval ligation in rats provided the first piece of evidence implicating the renal renin-angiotensin system in thirst, this conclusion has been questioned by several investigators (Lehr et al. 1975, Stricker 1977, Rolls & Wood 1977). The role of AII in drinking following water deprivation has also received much attention. Therefore, the role of the endogenous circulating AII in the control of drinking following water deprivation and caval ligation in rats is to be examined in Chapter VII.

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CHAPTER II GENERAL METHODS

This chapter describes the general methods used in Chapter III to VI. To avoid the complications of anesthesia, all the experiments were carried out on conscious animals.

Animals

Trained adult mongrel dogs of either sex were used throughout the studies. They were housed individually in an air-conditioned room maintained at 22°C and 55% humidity and on a 6 A.M. - 6 P.M. light cycle. They were fed a ration of dry Purina dog chow daily at 2 P.M. and allowed water ad libitum. The average daily sodium intake was approximately 90 mEq. On the day of experiment, the dog was brought into a quiet room and was minimally restrained in a dog sling (Alice King Chatham, Los Angeles) during the experiment.

Angiotensin II

Synthetic angiotensin II ($[\text{Asp}^1\text{-Ile}^5]$ angiotensin II, Bachem) was freshly prepared in a 0.9% NaCl solution to the concentration desired. It was infused at a

rate of 0.3 - 0.5 ml/min into an artery or 0.5 - 1.0 ml/min into a femoral vein for 10 min. The order of infusions was randomized, and consecutive infusions were separated by at least 10 min apart.

Surgical Procedures

A small non-occluding catheter was made of two pieces of tygon tubing (Norton) glued together with cyclohexanone: a 5-cm-long smaller tubing (0.75 mm I.D., 1.2 mm O.D.) inserted into a 90-cm-long larger tubing (1.1 mm I.D., 1.8 mm O.D.). After the dog was anesthetized with pentobarbital, a carotid or vertebral artery was occluded temporarily for inserting the smaller end of a non-occluding catheter. The catheter was then secured in place by a purse-string suture in the arterial wall (Rudolph et al. 1956).

A tygon tubing (1.3 mm I.D., 2.3 mm O.D.) was inserted into one end of the femoral artery and vein and advanced into the aorta and vena cava, respectively. The other end of the vessel was tied off.

All the catheters were filled with heparin, passed subcutaneously and exteriorized at the back of the dog. A dog jacket (Alice King Chatham) was worn to protect

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The dogs were allowed five to seven days to recover from the surgery. Their rectal temperatures were monitored daily and antibiotics (penicillin G and tylosin) were given if necessary.

Measurement of Cardiovascular Variables

The arterial blood pressure was measured by the femoral arterial catheter connected to a pressure transducer (P23Db, Statham). Mean arterial pressure was obtained by damping the arterial pressure electronically. Heart rate was measured by a tachometer, triggered by the arterial pulse pressure, and was checked by counting pulse waves in the arterial pressure tracing. All these variables were recorded on paper by a polygraph (Grass).

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CHAPTER III PRESSOR RESPONSE TO INTRACAROTID INFUSION
 OF ANGIOTENSIN II

INTRODUCTION

In addition to the systemic vasoconstrictor effect, blood-borne angiotensin II has a central pressor component (Bickerton & Buckley 1961). Administration of AII into the vertebral arteries has been shown to cause a centrally-mediated pressor response in rabbits (Yu & Dickson 1965), dogs (Lowe & Scroop 1969, Ferrario et al. 1970) and man (Ueda et al. 1969). The area postrema, a circumventricular organ in the caudal medulla, is generally taken as the site of action of this pressor effect of AII in dogs (Joy & Lowe 1970b, Gildenberg et al. 1973).

However, there is evidence that more rostral sites may also be involved in the central pressor action of AII. Intraventricular AII was shown to act on the sub-nucleus medialis in the midbrain to increase blood pressure in cats (Deuben & Buckley 1970). Transection of the midbrain eliminated the pressor response to intraventricular, but not intravertebral, AII in dogs (Gildenberg et al. 1973). Microinjection of AII into the subfornical organ, a circumventricular organ adja-

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cent to the third ventricle, increased blood pressure in rats (Mangiapane & Simpson 1980). Infusion of AII into the carotid artery, but not the vertebral artery, elicited a greater increase in blood pressure than intra-aortic AII did in rats (Haywood et al. 1980). This pressor effect was abolished by lesion of the anteroventral third ventricle (Fink et al. 1980), but not the area postrema (Haywood et al. 1980).

Although intracarotid AII was generally reported to have no central pressor action in dogs (Severs & Daniels-Severs 1973), most of the studies were done on anesthetized dogs (Lowe & Scroop 1969, Ferrario et al. 1970a, Joy & Lowe 1970). More recently Reid (1980) showed that infusion of AII into the common carotid arteries in conscious dogs induced a pressor response at doses that were ineffective when given intravenously. This pressor response differed from that produced by intravertebral AII in that it was smaller in magnitude, slower in onset, and not accompanied by an increase in heart rate. It could be mediated centrally; however, there exists at least one alternative.

Most of the blood flow in the common carotid artery perfuses the extracranial vascular beds via the external carotid artery in dogs. Being a potent

The first part of the report deals with the general situation of the country and the position of the various regions. It is followed by a detailed description of the economic and social conditions in each of the regions. The report concludes with a summary of the main findings and a list of recommendations.

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The third part of the report deals with the specific details of the economic and social conditions in each of the regions. It is followed by a detailed description of the economic and social conditions in each of the regions. The report concludes with a summary of the main findings and a list of recommendations.

vasoconstrictor, AII infused into the carotid artery is likely to significantly reduce the blood flow through the external carotid artery. This reduction in local blood flow could cause a pressor response. The possibility then exists that the pressor response to intracarotid AII might be simply due to extracranial vasoconstriction.

To test this hypothesis, carotid blood flow was monitored during intracarotid AII infusion. Then the pressor effect of AII infused into the common carotid arteries was compared with that of AII infused into the external carotid arteries. A theoretical analysis is presented in the Appendix to examine the quantitative relationship between the reduction in local blood flow and the pressor effect.

METHODS

1. Measurement of Carotid Blood Flow

A non-occluding catheter was chronically implanted in both common carotid arteries in 4 dogs (19-29 kg) for AII infusion. A pre-calibrated electromagnetic flow transducer (3-4 mm I.D., Biotronex) and a hydraulic silastic cuff (IVM, Healdsburg, CA) were implanted around the right common carotid artery for flow measurement. The cuff was positioned more distal

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from the heart than the flow transducer and was used to occlude the carotid blood flow for zero-flow determination.

After recovering from the surgery, the dog was brought into the laboratory and the flow transducer connected to a flowmeter (BL-10, Biotronex). After the dog had calmed down, AII (2.0 ng/kg/min) was infused bilaterally into common carotid arteries for 10 min. Carotid blood flow was monitored throughout the experiment.

2. Pressor Responses to Angiotensin II

A non-occluding catheter was chronically implanted in both common and both external carotid arteries in 7 dogs (17-25 kg) for AII infusions. To cannulate the external carotid artery, a catheter was inserted into the common carotid artery just below the carotid sinus bifurcation, and its tip was advanced beyond the bifurcation of the lingual artery from the external carotid artery. A femoral artery and vein were also cannulated for blood pressure measurement and AII infusion, respectively.

After recovering from the surgery, the dog was infused with AII into both common or both external carotid arteries or a femoral vein in a random order.

The first part of the report deals with the general situation of the country and the progress of the work done during the year. It is followed by a detailed account of the work done in each of the various departments. The report concludes with a summary of the work done and a statement of the results achieved.

The work done during the year has been very satisfactory and has resulted in a number of important discoveries. The most important of these are the discovery of the new element, the discovery of the new compound, and the discovery of the new process.

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Two doses of AII were infused: 1.0 and 2.0 ng/kg/min. Arterial blood pressure and heart rate were monitored throughout the experiment.

3. Data Analysis

Student's t-test for paired data was used to analyze carotid flow data. Analysis of variance for repeated measurements (Winer 1971) followed by Newman-Keuls post-hoc comparisons (Zar 1974) was used to analyze the pressor responses to different routes of AII infusion. The level of significance was set at 0.05. Data are expressed as mean±standard deviation.

RESULTS

The mean blood flow in the common carotid arteries of 4 dogs was 139 ± 26 ml/min during the control pre-infusion period. It decreased gradually and reached a mean of 71 ± 14 ml/min during intracarotid infusion of AII (Fig.III.1 & 2). This decrease was significant ($p < 0.05$).

The mean arterial pressure during the control pre-infusion period was approximately 100 mmHg. The increase in mean arterial pressure during AII infusion into the common carotid arteries was similar to that during AII infusion into the external carotid arteries,

1. The first part of the report deals with the general situation in the country and the results of the survey. It is divided into three sections: (a) general situation, (b) results of the survey, and (c) conclusions.

2. The second part of the report deals with the detailed results of the survey. It is divided into two sections: (a) results of the survey, and (b) conclusions.

3. The third part of the report deals with the detailed results of the survey. It is divided into two sections: (a) results of the survey, and (b) conclusions.

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and both were greater than that during intravenous AII at two different doses (Fig.III.3 & Table III.1). Heart rate did not change significantly during any routes of AII infusion (Table III.2).

Based on the assumption that the cardiac output and the total resistance of other vascular beds are unchanged, the pressor response induced by intracarotid AII can be predicted from Fig.III.4. For a typical ratio of cardiac output to bilateral carotid flows (r) of 10, halving bilateral carotid flows will cause a pressor effect of approximately 5 mmHg, provided the control pre-infusion mean aortic pressure is 100 mmHg.

DISCUSSION

Reid's (1980) recent finding that intracarotid AII induced a greater pressor response than AII i.v. in conscious dogs was confirmed in this study (Fig.III.3). However, previous studies on anesthetized dogs reported that the pressor response to intracarotid AII was no greater than that to AII i.v. (Lowe & Scroop 1969, Ferrario et al. 1970, Joy & Lowe 1970a). It is difficult to explain this discrepancy. However, different animal preparations could account for it. Sweet et al. (1971) reported that infusion of AII at a rate of 10 ng/kg/min into the vertebral artery, but not the common carotid

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artery, increased blood pressure in conscious dogs. However, the same dose of AII was also ineffective when given i.v. Their dogs appeared to be insensitive to AII since this dose of AII i.v. has otherwise been shown to be pressor in conscious dogs (Chapter VI, Fukiyama et al. 1971, Ramsay et al. 1978).

On the other hand, Fitzsimons et al. (1978) reported that intracarotid AII at a dose of 2-3 ng/kg/min increased blood pressure by approximately 10 mmHg in 2 conscious dogs, and that the same dose given i.v. was ineffective in 1 dog. In spite of the small sample size, they did statistics on these data. It is therefore unjustified for them to conclude that intracarotid AII induced no significant pressor response.

Angiotensin II infused into the renal artery and mesenteric vein has been shown to cause smaller pressor responses than AII i.v. (Akinkugbe et al. 1966) as the kidney and liver inactivate significant amounts of AII (Hodge et al. 1967). In contrast, even though as much as 50% of the AII administered into the carotid arteries was inactivated in its first passage through the head (Hodge et al. 1967, Reid, unpublished observation), intracarotid AII elicited a greater pressor response than AII i.v. (Fig.III.3). The question arises then: what is the mechanism of the pressor

action of intracarotid AII ?

Intracarotid AII could act on the brain to cause a centrally-mediated pressor effect. Since the carotid blood does not perfuse the hindbrain and AII does not cross the blood-brain barrier, AII must be acting on some circumventricular organ rostral to the area postrema. The subfornical organ (Mangiapane & Simpson 1980) and the organum vasculosum of the lamina terminalis (Fink et al. 1980), both adjacent to the third ventricle, are likely candidates since both have been proposed to contain receptors for pressor action of AII in rats.

Another alternative is that intracarotid AII could constrict the extracranial vascular beds to increase the systemic vascular resistance and result in a pressor response. When AII was infused at a rate of 1 ng/kg/min into each common carotid artery, the plasma AII concentration in the artery could reach over 200 pg/ml, approximately ten times normal concentration (Ramsay et al. 1978). Since AII is a potent vasoconstrictor and 90% of the common carotid blood flow perfuses the extracranial vascular beds (Chapter IV), it is not surprising to see that the blood flow in the common carotid artery halved during intracarotid AII infusion (Fig.III.2). This is in agreement with

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Greenfield & Tindall (1968), who reported that the external carotid blood flow reduced to two thirds of its control value shortly after an AII injection into the external carotid artery in one man. According to the theoretical analysis presented in the Appendix, this reduction in flow could result in a pressor response of 5 mmHg (Fig. III.4). which happened to be the difference in the pressor effects of intracarotid and intravenous AII (Fig. III.3). Therefore, the extracranial vasoconstriction hypothesis appears to be able to account for most of the pressor response to intracarotid AII, at least theoretically.

The extracranial vasoconstriction hypothesis would gain strong support from the result that external carotid infusion of AII increased blood pressure to the same degree as common carotid infusion did (Fig. III.3), provided that the external carotid blood did not perfuse the brain. However, Jewell (1952) has provided anatomic evidence that anastomoses exist between the external and internal carotid circulations in dogs. Before the functional significance of these anastomoses in conscious dogs is understood, which is the topic of the following chapter, the results of the present study do not permit definite conclusion concerning the mechanism of the pressor response to intracarotid AII.

1. The first part of the document is a letter from the author to the editor of the journal, dated 1st January 1971. The letter is addressed to the Editor, Journal of the Royal Society of Medicine, and is signed by the author, Dr. J. H. G. [Name]. The letter discusses the author's interest in the journal and the possibility of contributing to it. The author mentions that they have been reading the journal for some time and are impressed by the quality of the articles. They express a desire to contribute to the journal and ask if there are any opportunities for this. The letter is dated 1st January 1971 and is signed by Dr. J. H. G. [Name].

2. The second part of the document is a letter from the editor to the author, dated 15th January 1971. The letter is addressed to Dr. J. H. G. [Name] and is signed by the Editor, Journal of the Royal Society of Medicine. The letter responds to the author's letter and expresses interest in their work. The editor mentions that they have read the author's letter and are impressed by the author's interest in the journal. They mention that they are currently looking for new contributors and that the author's work would be a valuable addition to the journal. The editor offers the author the opportunity to submit an article to the journal and asks if they would like to do this. The letter is dated 15th January 1971 and is signed by the Editor, Journal of the Royal Society of Medicine.

APPENDIX

To derive the relationship between the change in mean aortic blood pressure and that in mean blood flow in some systemic artery,

let Pa = mean aortic blood pressure

Pv = mean vena caval blood pressure

CO = mean cardiac output

Qc = mean blood flow in some systemic artery

TPR = mean total peripheral resistance

$$= (Pa - Pv) / CO$$

Rc = total resistance of the local vascular beds perfused by the artery

$$= (Pa - Pv) / Qc$$

Ro = total resistance of all other systemic vascular beds

$$= (Pa - Pv) / (CO - Qc)$$

By definition,

$$Pa - Pv = CO \times TPR \quad \langle \text{III.1} \rangle$$

$$1/Rc + 1/Ro = 1/TPR \quad \langle \text{III.2} \rangle$$

In a new state, these equations become

$$Pa' - Pv' = CO' \times TPR' \quad \langle \text{III.1a} \rangle$$

$$1/Rc' + 1/Ro' = 1/TPR' \quad \langle \text{III.2a} \rangle$$

$\langle \text{III.2} \rangle - \langle \text{III.2a} \rangle$

1957-58

1. The first part of the problem is to find

the value of λ such that the system of linear equations

$$\begin{aligned}
x + y + z &= 1 \\
x + 2y + 3z &= \lambda \\
x + 3y + 4z &= 2
\end{aligned}$$

has a unique solution. For this purpose we write

$$\begin{aligned}
\Delta &= \begin{vmatrix} 1 & 1 & 1 \\ 1 & 2 & 3 \\ 1 & 3 & 4 \end{vmatrix} = 1(2 \cdot 4 - 3 \cdot 3) - 1(4 - 3) + 1(4 - 6) \\
&= 1(8 - 9) - 1(1) + 1(-2) = -1 - 1 - 2 = -4
\end{aligned}$$

Since $\Delta \neq 0$, the system has a unique solution for all values of λ .

$$\Delta_x = \begin{vmatrix} 1 & 1 & 1 \\ 1 & 2 & 3 \\ 1 & 3 & 4 \end{vmatrix} = 1(2 \cdot 4 - 3 \cdot 3) - 1(4 - 3) + 1(4 - 6) = -4$$

Similarly, $\Delta_y = \begin{vmatrix} 1 & 1 & 1 \\ 1 & 2 & 3 \\ 1 & 3 & 4 \end{vmatrix} = 1(2 \cdot 4 - 3 \cdot 3) - 1(4 - 3) + 1(4 - 6) = -4$

$$\Delta_z = \begin{vmatrix} 1 & 1 & 1 \\ 1 & 2 & 3 \\ 1 & 3 & 4 \end{vmatrix} = 1(2 \cdot 4 - 3 \cdot 3) - 1(4 - 3) + 1(4 - 6) = -4$$

Therefore,

$$x = \frac{\Delta_x}{\Delta} = \frac{-4}{-4} = 1$$

$$y = \frac{\Delta_y}{\Delta} = \frac{-4}{-4} = 1$$

and $z = \frac{\Delta_z}{\Delta} = \frac{-4}{-4} = 1$.

$$\therefore \text{The unique solution is } x = 1, y = 1, z = 1.$$

$$\text{Hence, the value of } \lambda \text{ is } 1 + 2 + 3 = 6.$$

$$(1/R_c - 1/R_c') + (1/R_o - 1/R_o') = (1/TPR - 1/TPR')$$

or

$$(1 - R_c/R_c') + (1/R_o - 1/R_o')R_c = (R_c/TPR)(TPR' - TPR)/TPR' \\ = rX/(1+X) \quad \langle \text{III.3} \rangle$$

where $TPR' = (1+X)TPR$

and $r = R_c/TPR = CO/Q_c$

Assume $R_o' \cong R_o$,

i.e., change in total resistance of all other systemic vascular beds is negligible,

then $\langle \text{III.3} \rangle$ becomes

$$1 - R_c/R_c' \cong rX/(1+X)$$

or

$$R_c'/R_c \cong (1+X)/[1-(r-1)X] \quad \langle \text{III.4} \rangle$$

Assume $CO' \cong CO$,

i.e., change in cardiac output is negligible,

then $\langle \text{III.1a} \rangle / \langle \text{III.1} \rangle$ becomes

$$(P_a' - P_v') / (P_a - P_v) \cong TPR' / TPR \\ \cong 1+X \quad \langle \text{III.5} \rangle$$

By definition,

$$Q_c = (P_a - P_v) / R_c$$

$$Q_c' = (P_a' - P_v') / R_c'$$

so $Q_c'/Q_c \cong (1+X)(R_c/R_c')$

$\langle \text{III.4} \rangle$ becomes

$$V(t) = \dots$$

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$$Q_c'/Q_c \approx 1-(r-1)X \quad \langle \text{III.4a} \rangle$$

Since $P_v, P_v' \ll P_a, P_a'$,

$\langle \text{III.5} \rangle$ becomes

$$P_a'/P_a \approx (P_a' - P_v') / (P_a - P_v) \approx 1+X$$

or

$$p/P_a \approx X$$

where

$$p = P_a' - P_a$$

$\langle \text{III.4a} \rangle$ becomes

$$Q_c'/Q_c \approx 1-(r-1)(p/P_a)$$

or

$$p/P_a \approx [1-(Q_c'/Q_c)] / (r-1) \quad \langle \text{III.6} \rangle$$

Therefore, when Q_c is changed to Q_c' , without concurrent changes in R_o and CO , the corresponding change in mean aortic pressure (p) can be predicted from $\langle \text{III.6} \rangle$. The straight lines plotted in Fig.III.4 represent Eq. $\langle \text{III.6} \rangle$ with different values of r .

Typically, $P_a \approx 100$ mmHg, and for both carotid arteries, $r \approx 10$, to induce a pressor response of 5 mmHg, $Q_c' \approx 0.55 Q_c$, i.e., one can increase blood pressure by 5 mmHg by reducing carotid blood flows to half.

$$f(x) = \frac{1}{2} (e^x + e^{-x})$$

where $f(x)$ is the function defined by

$$f(x) = \frac{1}{2} (e^x + e^{-x})$$

$$f'(x) = \frac{1}{2} (e^x - e^{-x})$$

$$f''(x) = \frac{1}{2} (e^x + e^{-x})$$

$$f'''(x) = \frac{1}{2} (e^x - e^{-x})$$

$$f^{(4)}(x) = \frac{1}{2} (e^x + e^{-x})$$

we

$$\langle f, f \rangle = \int_{-\infty}^{\infty} f(x) f(x) dx = \int_{-\infty}^{\infty} \frac{1}{4} (e^x + e^{-x})^2 dx$$

... the integral ...

$$\int_{-\infty}^{\infty} \frac{1}{4} (e^{2x} + 2 + e^{-2x}) dx = \dots$$

... the result ...

The above derivation was only based on two assumptions, i.e., $Ro' \approx Ro$ and $CO' \approx CO$. When a vasoconstrictor such as AII is infused into the artery, Ro' and CO' are more likely to be less than Ro and CO , respectively, due to the baroreceptor reflex.

Then Eq.<III.5> becomes

$$Pa'/Pa \leq TPR'/TPR = 1+X$$

or

$$p/Pa \leq X$$

Eqs.<III.4a> and <III.6> become

$$Qc'/Qc \leq 1-(r-1)X$$

and

$$p/Pa \leq X \leq [1-(Qc'/Qc)]/(r-1)$$

Therefore, Eq.<III.6> overestimates p/Pa ; the actual lines in Fig. III.4 will shift downward. However, if peripheral spill-over of the vasoconstrictor occurs, i.e., the vasoconstrictor acts on other systemic beds as well, then the lines will shift upward.

Finally, Eq.<III.6> applies to not only vasoconstrictors but also vasodilators, where $Qc' > Qc$ and $p < 0$.

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Table III.1 Mean arterial blood pressure (mmHg) during the control pre-infusion periods and its changes during infusions of angiotensin II (AII) at two doses into both common carotid arteries (CC), both external carotid arteries (EC), and the femoral vein (iv).

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(a) AII (2 x 1.0 ng/kg/min)

Dog	Control	CC AII	Control	EC AII	Control	iv AII
1	109	+11	106	+10	112	+5
2	125	+15	118	+18	125	+5
3	125	+16	101	+14	107	+11
4	88	+14	97	+8	104	+6
5	90	+8	89	+8	91	+6
6	83	+9	83	+10	87	+4
7	78	+5	78	+3	80	+8
mean	96	+11	96	+10	101	+6
SD	16	4	14	5	16	2

(b) AII (2 x 0.5 ng/kg/min)

1	100	+6	100	+5	100	+3
2	130	+8	125	+11	124	+2
3	105	+10	107	+8	109	+4
4	103	+7	97	+10	104	+3
5	85	+4	85	+5	83	+2
6	83	+3	83	+4	82	+1
7	80	+2	80	+3	82	+5
mean	98	+6	97	+7	98	+3
SD	17	3	16	3	16	1

PLATE 1. 1953-54. (continued)

	1953	1954	1955	1956	1957	1958
1.	1.1	1.1	1.1	1.1	1.1	1.1
2.	2.1	2.1	2.1	2.1	2.1	2.1
3.	3.1	3.1	3.1	3.1	3.1	3.1
4.	4.1	4.1	4.1	4.1	4.1	4.1
5.	5.1	5.1	5.1	5.1	5.1	5.1
6.	6.1	6.1	6.1	6.1	6.1	6.1
7.	7.1	7.1	7.1	7.1	7.1	7.1
8.	8.1	8.1	8.1	8.1	8.1	8.1
9.	9.1	9.1	9.1	9.1	9.1	9.1
10.	10.1	10.1	10.1	10.1	10.1	10.1

PLATE 2. 1953-54.

	1953	1954	1955	1956	1957	1958
1.	1.1	1.1	1.1	1.1	1.1	1.1
2.	2.1	2.1	2.1	2.1	2.1	2.1
3.	3.1	3.1	3.1	3.1	3.1	3.1
4.	4.1	4.1	4.1	4.1	4.1	4.1
5.	5.1	5.1	5.1	5.1	5.1	5.1
6.	6.1	6.1	6.1	6.1	6.1	6.1
7.	7.1	7.1	7.1	7.1	7.1	7.1
8.	8.1	8.1	8.1	8.1	8.1	8.1
9.	9.1	9.1	9.1	9.1	9.1	9.1
10.	10.1	10.1	10.1	10.1	10.1	10.1

Table III.2 Mean heart rate (beats/min) during control pre-infusion periods and its changes during infusions of AII at two doses into both common carotid arteries, both external carotid arteries, and the femoral vein.

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(a) AII (2 x 1.0 ng/kg/min)

Dog	Control	CC AII	Control	EC AII	Control	iv AII
1	55	-1	55	-4	52	+2
2	55	0	70	-5	55	-5
3	45	-7	48	-10	45	0
4	72	-7	69	-7	66	0
5	67	-1	58	-1	56	+1
6	83	+3	83	+4	82	+1
7	89	-9	94	-14	91	-1
mean	67	-3	68	-5	64	0
SD	16	4	16	6	17	2

(b) AII (2 x 0.5 ng/kg/min)

1	56	0	56	-3	54	-2
2	68	+2	60	+7	63	-9
3	45	-5	55	-5	45	-2
4	63	+5	60	0	52	+5
5	77	+3	78	-2	78	0
6	55	-1	58	-3	65	-6
7	80	+1	90	-4	97	+3
mean	63	+1	65	-1	65	-2
SD	13	3	13	4	18	5

QUESTION

QUESTION	ANSWER	QUESTION	ANSWER
1	-	11	+
2	-	12	-
3	-	13	-
4	-	14	-
5	-	15	-
6	-	16	-
7	-	17	-
8	-	18	-
9	-	19	-
10	-	20	-

QUESTION

1	-	11	+
2	-	12	+
3	-	13	-
4	+	14	+
5	-	15	-
6	-	16	-
7	-	17	+
8	-	18	-
9	+	19	+
10	-	20	+

Fig. III.1 A typical example of the effects of intracarotid AII on arterial blood pressure (mmHg) and mean blood flow (ml/min) in the right common carotid artery (CC) of a conscious dog. Angiotensin was infused at 1 ng/kg/min into each artery. The left arrow indicates the beginning of AII infusion, and the right one the end of infusion. MAP: mean arterial blood pressure. BP: arterial blood pressure.

Fig. III.1

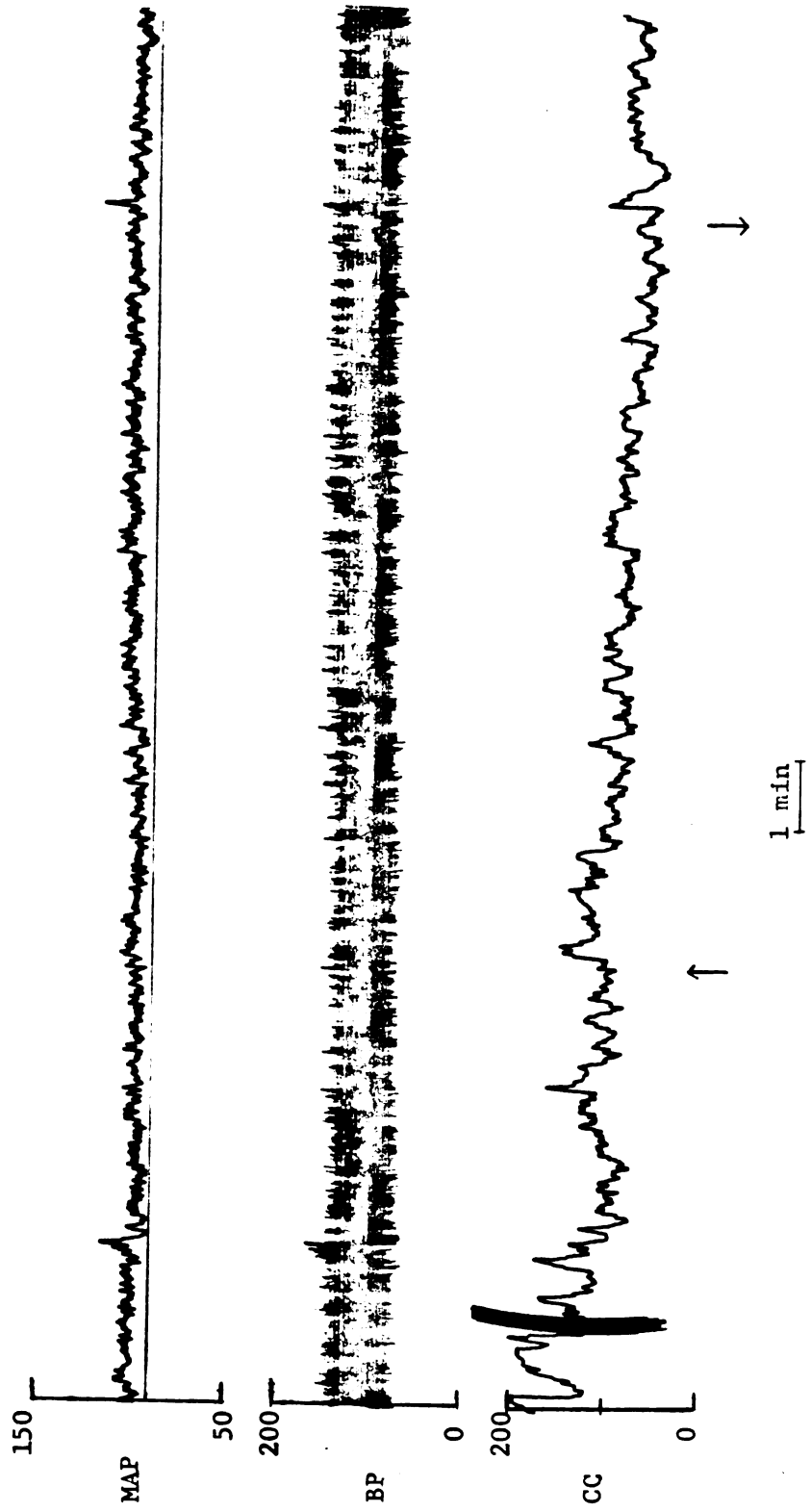


Fig. III.2 Mean blood flow (ml/min) in the right common carotid artery during the control pre-infusion period and intracarotid AII infusion (C.C.AII) in 4 dogs. Different symbols represent different dogs. The dose of AII is explained in the previous figure.

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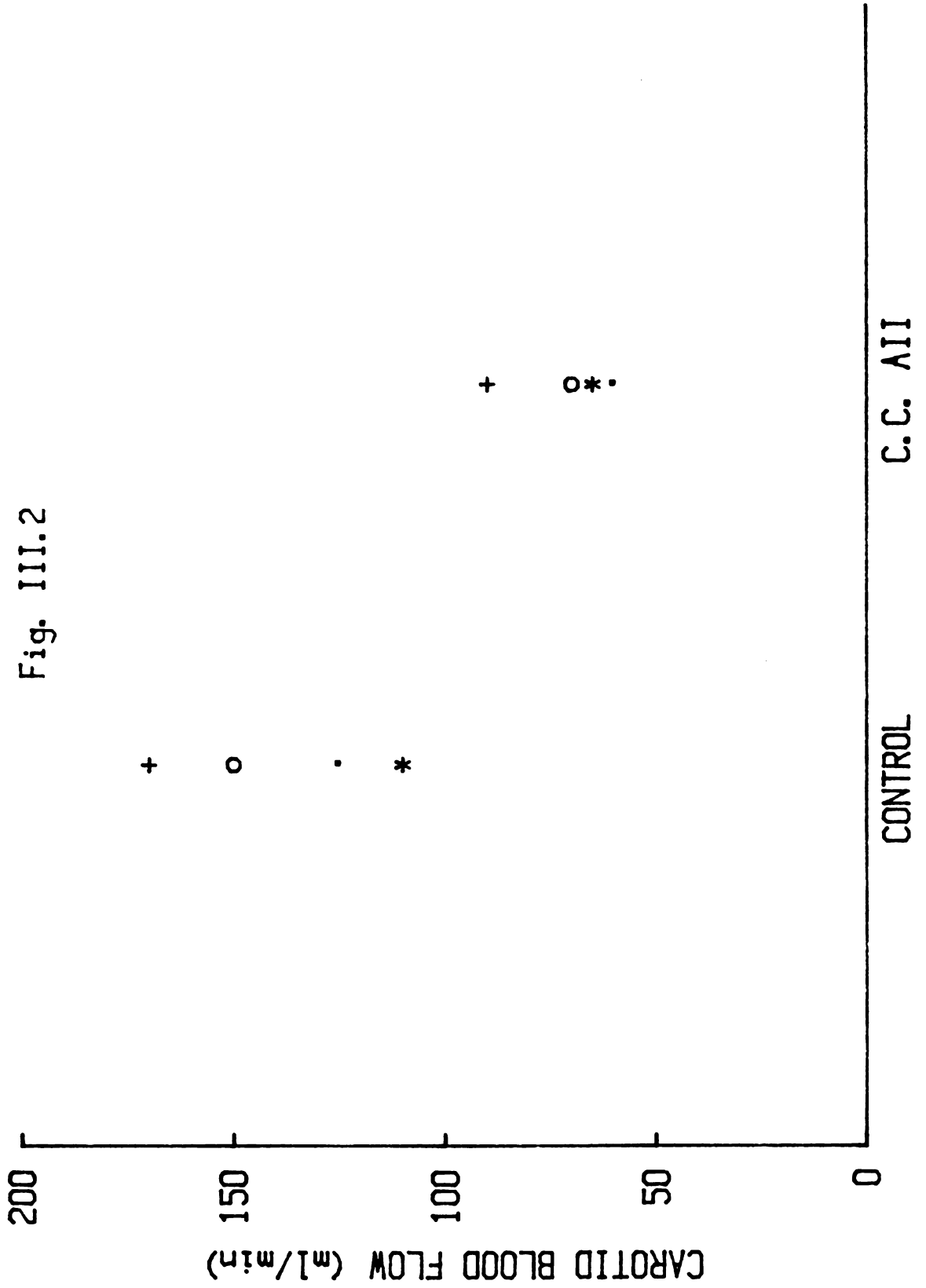


Fig. III.3 Mean pressor response during AII infusions into both common carotid arteries (CC), both external carotid arteries (EC) and the femoral vein (i.v.) at two doses in 7 dogs. * significantly different from i.v. ($p < 0.05$). mean_±SEM are shown.

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Fig. III.3

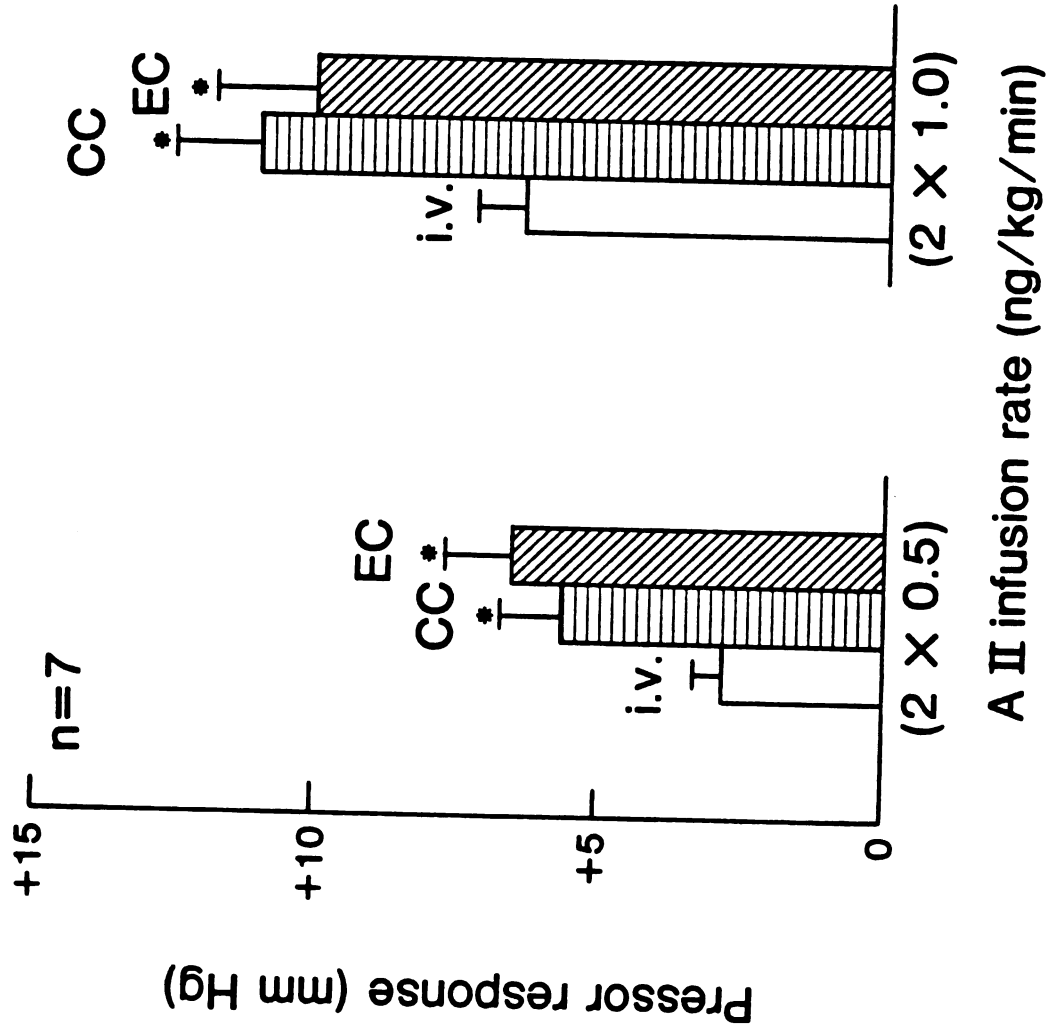


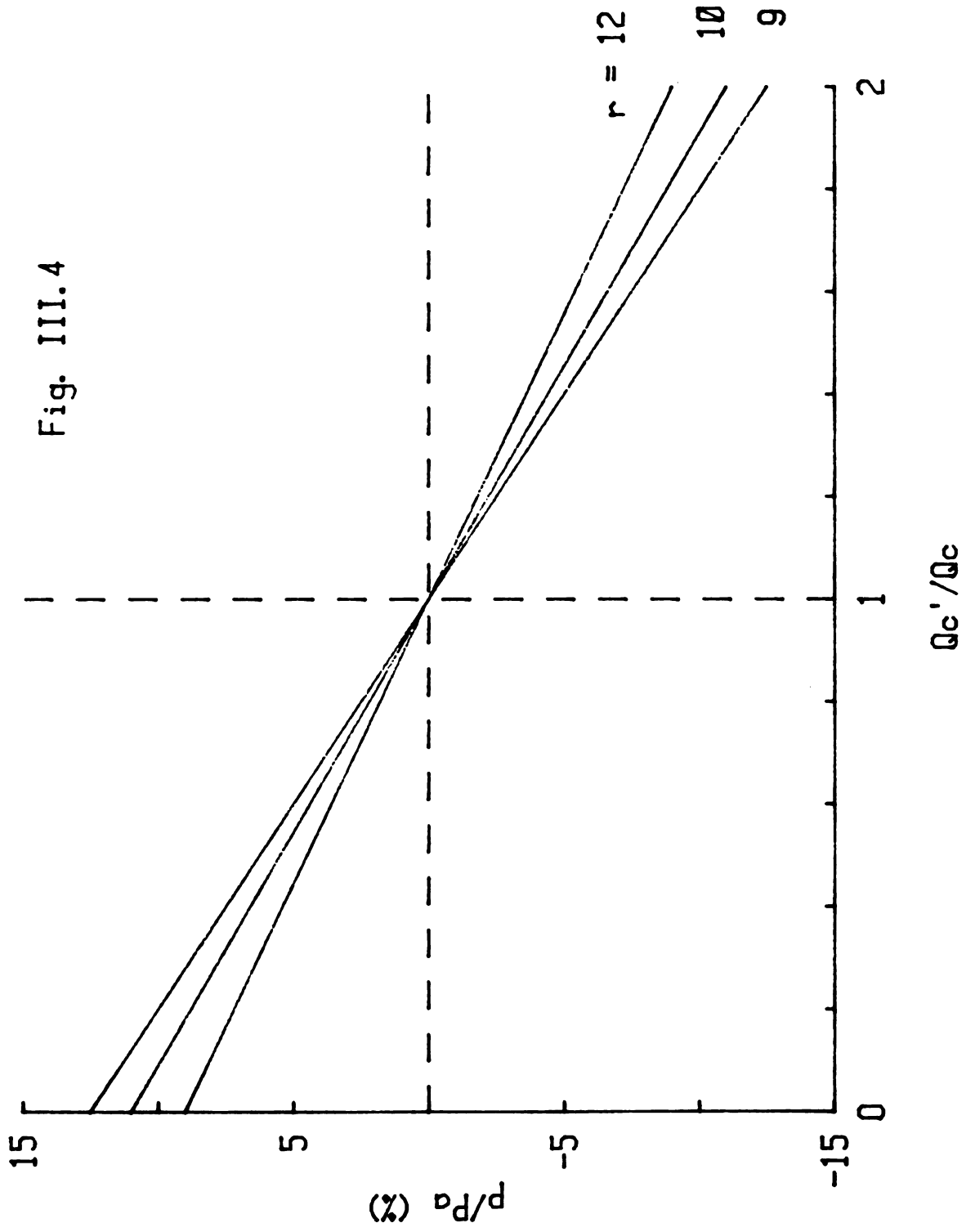
Fig. III.4 Change in mean aortic pressure (p) vs blood flow in some systemic artery (Q_c') as predicted from Eq.<III.6>. Different lines represent different ratios of cardiac output to the control blood flow in the artery (r). P_a : control mean aortic pressure. Q_c : control mean blood flow in the artery.

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CHAPTER IV ANASTOMOSES BETWEEN THE EXTERNAL CAROTID
AND INTRACRANIAL CIRCULATIONS

INTRODUCTION

Several anastomoses exist between the external carotid and intracranial circulations in dogs, through which external carotid blood can perfuse the brain. The anastomotic artery running between the external ophthalmic and internal carotid arteries was described long ago by Ellenberger & Baum (1891) and its functional importance was studied by Bouckaert & Heymans (1935). Anatomic evidence on other anastomoses between the external carotid and intracranial circulations in dogs was described in detail by Jewell (1952). Angiographic studies on these anastomoses were reported by de la Torre et al. (1959). However, these studies provided only qualitative descriptions on the anastomoses; moreover, none were done on conscious dogs.

To provide quantitative data on these anastomoses, in the present study radioactive microspheres were injected into the common and external carotid arteries of conscious dogs. By comparing microsphere distributions in the brain and extracranial vasculature, the blood flows in the anastomoses and internal carotid

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artery were determined. To study the functional significance of the anastomoses, the effects of infusions of angiotensin II into the common and external carotid arteries on vasopressin and corticosteroid secretion were compared.

METHODS

Microsphere Studies

Four dogs (17-24 kg) were chronically prepared with non-occluding catheters in common and external carotid arteries on both sides for microsphere injections. The tip of the common carotid catheter pointed toward the heart to assure a good mixing of the injected spheres within the blood stream.

Fifteen μm radioactive spheres (3M) labeled with Gd^{153} , Co^{57} , Sn^{113} , Sr^{85} , Mn^{54} , and Zn^{65} were used in this study. The spheres were suspended in a 5% dextran solution with 0.05% polyoxyethylene 80 sorbitan monooleate (Tween 80) to prevent aggregation (Heymann et al. 1977). Approximately half a million spheres were placed in a 0.5-ml injector vial. After vigorous shaking of the vial, 2.5 ml of saline was flushed through the vial into the catheter over 30 s. The number of spheres injected was determined by counting the radioactivity of the vial before and after the

The first part of the report deals with the general situation of the country and the results of the survey. It is followed by a detailed description of the various types of land use and the distribution of the population. The third part of the report is devoted to a study of the economic situation and the social conditions of the population. The fourth part of the report contains a summary of the findings and a list of references.

1. General situation

The country is situated in the north-western part of the continent. It is a large country with a population of about 10 million. The climate is temperate and the soil is fertile. The main occupations of the population are agriculture and stock raising. The country has a long history and a rich cultural heritage. It is a member of the Commonwealth of Nations and the Organisation of African States.

The population is distributed unevenly over the country. The most densely populated areas are the coastal regions and the areas around the major cities. The rural population is engaged in agriculture and stock raising. The urban population is engaged in commerce and industry. The country has a high literacy rate and a growing economy. It is a member of the Commonwealth of Nations and the Organisation of African States. The country has a long history and a rich cultural heritage. It is a member of the Commonwealth of Nations and the Organisation of African States.

injection.

At least two different isotopes were injected into both common and both external carotid arteries of the dog. The isotopes and the order of injections were randomized.

During common carotid injections, a roller pump (Cole-Parmer, Chicago) was used to withdraw blood, at a rate of 1.5-2.5 ml/min, from the external carotid artery to estimate the blood flow in the common carotid artery. The pump was turned on 1 min before and remained on until 3 min after the sphere injection.

After the experiment, the dog was killed with an overdose of sodium pentobarbital. The brain as well as the masseter muscles was removed and fixed in 4% formaldehyde. Three to five days later, the brain was dissected into pieces and weighed. After carbonization, all the samples were counted for the radioactivity of each isotope by an Ultima/II multichannel pulse-height analyzer (Ino-Tech, Fort Atkinson, Wisconsin).

Blood Flow Estimation

Blood flow in the common carotid artery was estimated by the reference sample method (Heymann et

al. 1977), i.e., $\text{Flow} = (\text{reference sample withdrawal rate}) / (\text{fraction of injected spheres in the reference sample})$.

To estimate the anastomotic and internal carotid blood flows, let

CC = Common carotid blood flow

EC' = External carotid blood flow beyond the bifurcation of lingual artery

IC = Internal carotid blood flow

A = Anastomotic flow between the intracranial and external carotid circulations beyond the bifurcation of lingual artery

B, B' = Fractions of injected spheres trapped in the brain when injected into the common and external carotid arteries, respectively

T, T' = Fractions of injected spheres trapped in the masseter muscles when injected into the common and external carotid arteries, respectively

For tissues which are perfused by the external carotid artery, such as the masseter muscles (Evans & Christensen 1979),

$$\begin{aligned} \text{EC}' \times \text{T}' &= \text{Blood flow to masseter muscles} \\ &= \text{CC} \times \text{T} \end{aligned}$$

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$$EC' = CC \times T/T' \quad \langle IV.1 \rangle$$

When spheres were injected into the external carotid artery, the only routes that the spheres could reach the brain were via the anastomoses (Fig.IV.1), hence

$$B' = A / EC' \quad \langle IV.2 \rangle$$

When spheres were injected into the common carotid artery, they could reach the brain via both the internal carotid artery and the anastomoses, hence

$$B = (A + IC) / CC \quad \langle IV.3 \rangle$$

Since T, T', B, and B' were measured and CC was estimated by the reference sample method, EC' could be calculated from Eq.<IV.1>, anastomotic flow (A) from Eq.<IV.2>, and the internal carotid flow (IC) from Eq.<IV.3>.

Humoral Responses to Angiotensin II Infusion

Angiotensin II was infused into both common or both external carotid arteries or femoral vein in 7 dogs (17-25 kg) at a rate of 5.0 ng/kg/min for 10 min. Blood samples were taken just before and at the end of infusion for arginine vasopressin assay, and in 5 dogs for 11-hydroxycorticosteroids assay. Changes in the plasma concentration of corticosteroids were used as an index of changes in ACTH secretion.

11.112

(1) $\frac{1}{x^2} = x^{-2}$

Derivative of x^{-2} is $-2x^{-3}$ which is $-\frac{2}{x^3}$

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Plasma vasopressin concentration was determined by radioimmunoassay (Keil & Severs 1977). Plasma 11-hydroxycorticosteroids were determined by a competitive protein binding radioassay (Murphy 1967). In the dog, these consist mainly of cortisol plus some corticosterone.

To analyze the humoral responses to different routes of AII infusion, analysis of variance for repeated measurements followed by Newman-Keuls post-hoc comparisons was used. The level of significance was set at 0.05. Data are expressed as mean±standard deviation.

RESULTS

Microsphere Studies

During microsphere injections, the dog remained calm and the arterial blood pressure and heart rate were constant. When injected into the common carotid arteries, $8.3 \pm 2.6\%$ of the injected spheres reached the brain; so did $4.5 \pm 1.3\%$ of the spheres injected into the external carotid arteries (Tables IV.1 & 2). Very few spheres reached the brainstem and cerebellum, while more than 95% of the brain spheres were located in the telecephalon.

The first part of the report deals with the general situation in the country. It is noted that the economy is in a state of stagnation and that the government has failed to implement the necessary reforms. The report also mentions that the population is suffering from poverty and unemployment.

The second part of the report discusses the political situation. It is stated that the government is corrupt and that there is a lack of transparency in its operations. The report also mentions that there is a growing opposition to the government and that the country is heading towards a crisis.

The third part of the report focuses on the social situation. It is noted that there is a high level of inequality in the country and that the poor are being exploited by the rich. The report also mentions that there is a lack of access to basic services such as education and healthcare.

The fourth part of the report discusses the international situation. It is stated that the country is being isolated by the international community and that it is facing economic sanctions. The report also mentions that there is a need for international aid and support.

The mean blood flow in each common carotid artery estimated by the reference sample method was 140 ± 32 ml/min (Table IV.3). The calculated internal carotid blood flow ranged from 6.1 to 9.3 with a mean of 7.7 ml/min/artery, and the anastomotic flow ranged from 2.4 to 4.9 with a mean of 3.3 ml/min. The ratio of the anastomotic to internal carotid flow ranged from 0.3 to 0.6.

To study the sphere distributions in the brain, the telecephalons of 3 dogs were grossly divided into 3 parts of roughly equal weight: anterior, middle and posterior. The percentage of total spheres in the brain located in each part of the telecephalon when the spheres were injected into the external carotid artery was almost identical to that when injected into the common carotid artery (Fig.IV.2). In other words, microsphere distributions in the telecephalon were similar whether the spheres were injected into the external carotid or into the common carotid artery.

Humoral Responses to Angiotensin II

Plasma arginine vasopressin concentration in 7 dogs increased from a mean of 2.0 to 3.8 pg/ml during common carotid infusions of AII (Table IV.4). The vasopressin concentration increased only slightly from

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2.1 to 2.9 pg/ml after external carotid AII, and this increase was significantly less than that after common carotid AII ($p < 0.05$). Vasopressin concentration was unchanged after intravenous AII.

Plasma 11-hydroxycorticosteroids concentration in 5 dogs increased significantly from a mean of 2.2 to 4.9 and 2.3 to 4.9 $\mu\text{g}/\text{dl}$ after AII infusions into the common and external carotid arteries, respectively (Table IV.5). It decreased slightly from 2.6 to 2.0 $\mu\text{g}/\text{dl}$ after intravenous AII.

DISCUSSION

The present study confirmed the existence of and, more importantly, provided quantitative data on the anastomoses between the intracranial and external carotid circulations in conscious dogs.

The microsphere method used in this study is very reliable in estimating blood flows so long as all the requirements are met (Heymann et al. 1977).

Firstly, the injected microspheres must be well-mixed within the blood stream. Since the common carotid catheters pointed against the blood flow, the spheres injected through these catheters were likely to mix well within the blood stream. This view was sup-

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 65. Mr. K. V. Brown, 6262 Peach St., Tallahassee, Fla.

 66. Mr. L. W. White, 6363 Peach St., Tallahassee, Fla.

 67. Mr. M. X. Black, 6464 Peach St., Tallahassee, Fla.

 68. Mr. N. Y. Green, 6565 Peach St., Tallahassee, Fla.

 69. Mr. O. Z. Brown, 6666 Peach St., Tallahassee, Fla.

 70. Mr. P. A. White, 6767 Peach St., Tallahassee, Fla.

 71. Mr. Q. B. Black, 6868 Peach St., Tallahassee, Fla.

 72. Mr. R. C. Green, 6969 Peach St., Tallahassee, Fla.

 73. Mr. S. D. Brown, 7070 Peach St., Tallahassee, Fla.

 74. Mr. T. E. White, 7171 Peach St., Tallahassee, Fla.

 75. Mr. U. F. Black, 7272 Peach St., Tallahassee, Fla.

 76. Mr. V. G. Green, 7373 Peach St., Tallahassee, Fla.

 77. Mr. W. H. Brown, 7474 Peach St., Tallahassee, Fla.

 78. Mr. X. I. White, 7575 Peach St., Tallahassee, Fla.

 79. Mr. Y. J. Black, 7676 Peach St., Tallahassee, Fla.

 80. Mr. Z. K. Green, 7777 Peach St., Tallahassee, Fla.

 81. Mr. A. L. Brown, 7878 Peach St., Tallahassee, Fla.

 82. Mr. B. M. White, 7979 Peach St., Tallahassee, Fla.

 83. Mr. C. N. Black, 8080 Peach St., Tallahassee, Fla.

 84. Mr. D. O. Green, 8181 Peach St., Tallahassee, Fla.

 85. Mr. E. P. Brown, 8282 Peach St., Tallahassee, Fla.

 86. Mr. F. Q. White, 8383 Peach St., Tallahassee, Fla.

 87. Mr. G. R. Black, 8484 Peach St., Tallahassee, Fla.

 88. Mr. H. S. Green, 8585 Peach St., Tallahassee, Fla.

 89. Mr. I. T. Brown, 8686 Peach St., Tallahassee, Fla.

 90. Mr. J. U. White, 8787 Peach St., Tallahassee, Fla.

 91. Mr. K. V. Black, 8888 Peach St., Tallahassee, Fla.

 92. Mr. L. W. Green, 8989 Peach St., Tallahassee, Fla.

 93. Mr. M. X. Brown, 9090 Peach St., Tallahassee, Fla.

 94. Mr. N. Y. White, 9191 Peach St., Tallahassee, Fla.

 95. Mr. O. Z. Black, 9292 Peach St., Tallahassee, Fla.

 96. Mr. P. A. Green, 9393 Peach St., Tallahassee, Fla.

 97. Mr. Q. B. Brown, 9494 Peach St., Tallahassee, Fla.

 98. Mr. R. C. White, 9595 Peach St., Tallahassee, Fla.

 99. Mr. S. D. Black, 9696 Peach St., Tallahassee, Fla.

 100. Mr. T. E. Green, 9797 Peach St., Tallahassee, Fla.

ported by the result that the common carotid blood flow estimated by the reference sample technique (Table IV.3) was so close to that measured by an electromagnetic flowmeter as described in Chapter III (Fig.III.2). However, the external carotid injections of microspheres posed a problem since these catheters pointed along the flow. It was hoped that the presence of the catheter itself might be able to generate turbulence and that the tortuosity and length of the external carotid artery could result in adequate mixing of the spheres within the blood stream. The small variabilities in the estimated values of EC'/CC (Table IV.3) and in the brain microsphere distributions (Table IV.2) supported this view.

Secondly, the spheres must be large enough to be trapped in the microvessels in their first circulation after injection; however, they must be small enough to distribute evenly within the blood stream to reflect the actual blood flow pattern. Fifteen μm spheres used in this study have been generally thought to fit these criteria and recommended for studies in the cerebral blood flow (Marcus et al. 1976 & Fan et al. 1979). Although this might not be the case for studies in blood flow to the facial muscles due to the possible existence of large arteriovenous shunts, the ratio of

The first part of the report deals with the general situation in the country. It is noted that the economy is still in a state of stagnation and that the government has failed to implement the necessary reforms. The second part of the report discusses the political situation and the role of the opposition. It is stated that the opposition is weak and that the government is not accountable to the people. The third part of the report deals with the social situation and the living conditions of the population. It is noted that the majority of the population is poor and that there is a high level of unemployment. The fourth part of the report discusses the international situation and the role of the country in the world. It is stated that the country is isolated and that it has no friends. The fifth part of the report deals with the future of the country and the role of the people. It is stated that the people must take responsibility for their own future and that they must work for a better future.

The sixth part of the report discusses the role of the media and the role of the intellectuals. It is stated that the media is controlled by the government and that the intellectuals are not free to express their opinions. The seventh part of the report deals with the role of the military and the role of the police. It is stated that the military is not professional and that the police are corrupt. The eighth part of the report discusses the role of the judiciary and the role of the legal system. It is stated that the judiciary is not independent and that the legal system is not fair. The ninth part of the report deals with the role of the education system and the role of the teachers. It is stated that the education system is not of high quality and that the teachers are not well paid. The tenth part of the report discusses the role of the health system and the role of the doctors. It is stated that the health system is not of high quality and that the doctors are not well paid.

EC'/CC (or T/T') is unaffected by these shuntings because the same correction factor would appear in both T and T' and cancel out each other (Eq.<IV.1>).

Finally, the number of spheres injected must be large enough to give reliable counts (Buckberg et al. 1971); however, it must not be too large to affect general and local circulations. A million spheres were injected at a time, which result in more than a thousand spheres in each sample, more than enough to give reliable counts. On the other hand, these injections appeared not to affect circulations since the dogs remained calm and their blood pressures and heart rates were unchanged during the experiments. Therefore, all the requirements for microsphere studies seem well met in this study.

According to Jewell (1952), there are five anastomoses between the external carotid and intracranial circulations: the occipital-vertebral anastomosis, the ascending pharyngeal-internal carotid anastomosis, and the three anastomoses between the internal maxillary artery and intracranial vessels -- the anastomotic artery, the ophthalmic anastomosis, and the ethmoidal anastomosis.

In the analysis of anastomotic flows (Fig.IV.1),

the occipital-vertebral and ascending pharyngeal-internal carotid anastomoses were ignored for two reasons: Firstly, if the occipital-vertebral anastomosis is functionally important, one should expect to see substantial number of spheres in the brainstem and cerebellum when the spheres are injected into the common carotid artery. However, this is not so (Table IV.1), It is therefore concluded that this anastomosis is unlikely to be functionally important in normal conscious dogs. Secondly, although this study provided no information about the functional significance of the ascending pharyngeal - internal carotid anastomosis, Jewell(1952) and others concluded that in dogs this anastomosis was of little or no functional significance. Hence it seemed justified to ignore this anastomosis too.

Of the three anastomoses between the internal maxillary artery and intracranial vessels, the anastomotic artery is generally thought to be the most important one (Jewell 1952). Therefore, although the anastomotic flow (A) estimated in this study theoretically is the total flow of all the anastomoses beyond the bifurcation of the lingual artery, practically it could be taken as primarily the flow through the anastomotic artery alone. This view is supported by the finding

- The first part of the report is devoted to a general survey of the situation in the country. It is based on the information received from the various sources mentioned in the text. The second part of the report is devoted to a detailed analysis of the situation in the various regions of the country. It is based on the information received from the various sources mentioned in the text. The third part of the report is devoted to a detailed analysis of the situation in the various regions of the country. It is based on the information received from the various sources mentioned in the text.

- The fourth part of the report is devoted to a detailed analysis of the situation in the various regions of the country. It is based on the information received from the various sources mentioned in the text. The fifth part of the report is devoted to a detailed analysis of the situation in the various regions of the country. It is based on the information received from the various sources mentioned in the text.

that the microsphere distributions in the telecephalon were similar whether the spheres were injected into the common or external carotid artery (Fig.IV.2). This finding suggests that the anastomotic flow mixes well with the internal carotid flow before reaching the cerebral microcirculations. Of the above three anastomoses only the blood in the anastomotic artery is able to do so because only it joins the internal carotid artery before reaching the circle of Willis (Jewell 1952).

The blood flows in the internal carotid and anastomotic arteries estimated in this study seem reasonable when compared with previous reports. The internal carotid blood flow measured by an electromagnetic flowmeter when the external carotid artery was clamped in anesthetized dogs was 20 ml/min (Vidrio & Hong 1976), more than twice the present estimate (7.7 ml/min). This difference is expected since Schneider & Schneider (1934) reported that internal carotid blood flow doubled when the external carotid artery was ligated because of the existence of anastomoses between intra- and extra- cranial circulations (Bouckaert & Heymann 1935). The mean telecephalon blood flow calculated from dividing the sum of the internal carotid and anastomotic flows by the telecephalon weight (65 ± 6 g)

was 35 ± 10 ml/100 g/min in the present study. This flow rate is in agreement with previous studies (Roth et al. 1970, Fan et al. 1979, Marcus et al. 1981) although it underestimates the actual telecephalon blood flow since the basilar artery is believed to perfuse part of the telecephalon via the posterior cerebral artery (Jewell & Verney 1957, de la Torre et al. 1962, Wellens et al. 1975).

According to the present estimation, the anastomotic flow is about half of the internal carotid flow (Table IV.3), therefore the external carotid artery contributes significantly to the perfusion of the brain in normal conscious dogs. This point was supported by the result that external carotid infusion of AII increased plasma 11-hydroxycorticosteroids concentration, used as an index of ACTH secretion, to the same extent as common carotid infusion did (Table IV.5). However, 8.3% of the spheres injected into the common carotid artery reached the brain while only 4.5% did when injected into the external carotid artery (Tables IV.1 & 2). Hence, the brain received higher concentration of AII when AII was infused into the common carotid artery than when same dose of AII was infused into the external carotid artery. This probably explains why plasma vasopressin concentration increased more

The first part of the report deals with the general situation of the country and the position of the various groups. It is a very interesting and detailed study of the social and economic conditions of the country. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

The second part of the report deals with the political situation of the country. It discusses the various political parties and their policies. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

The third part of the report deals with the economic situation of the country. It discusses the various economic sectors and their contribution to the national income. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

The fourth part of the report deals with the social situation of the country. It discusses the various social problems and the measures taken to solve them. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

The fifth part of the report deals with the cultural situation of the country. It discusses the various cultural activities and the role of the arts and sciences. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

The sixth part of the report deals with the international situation of the country. It discusses the country's relations with other countries and its role in the world. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

The seventh part of the report deals with the future of the country. It discusses the various challenges facing the country and the measures that should be taken to meet them. The author has done a great deal of research and has gathered a wealth of material which is presented in a clear and concise manner. The report is well written and is a valuable contribution to the knowledge of the country.

when AII was infused into the common carotid artery than when AII was infused into the external carotid artery (Table IV.4). However, it is difficult to explain the difference in the vasopressin and corticosteroids responses to external carotid AII; further research is in order.

In conclusion, the anastomoses between the external carotid and intracranial circulations are functionally important in conscious dogs. Therefore, the possibility still exists that the pressor response to external carotid AII described in Chapter III is due to a central action of AII.

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Table IV.1 Distribution of microspheres in the brain when injected into the common carotid artery. Data are expressed as percentage of injected spheres.

Dog #	1	2	3	4	mean	SD
medulla	0.002	0.009	0.001	0.001	0.003	0.004
pons	0.002	0.002	0	0	0.001	0.001
midbrain	0.002	0.001	0.001	0	0.001	0.001
cerebellum	0.001	0.020	0.002	0.002	0.006	0.009
pituitary	0.052	0.018	0.014	0.050	0.034	0.020
diencephalon	0.080	0.71	0.038	0.058	0.22	0.33
telecephalon	8.18	7.52	5.03	11.26	8.00	2.56
whole brain	8.32	8.28	5.09	11.3	8.26	2.56

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Table IV.2 Distribution of microspheres in the
brain when injected into the external carotid artery.
Data are expressed as percentage of injected spheres.

Dog #	1	2	3	4	mean	SD
medulla	0.001	0.002	0	0	0.001	0.001
pons	0.002	0.002	0	0.002	0.002	0.001
midbrain	0.001	0.006	0	0	0.002	0.003
cerebellum	0.001	0.002	0.001	0	0.001	0.001
pituitary	0.027	0.020	0	0.001	0.012	0.014
diencephalon	0.080	0.35	0.013	0.002	0.11	0.16
telecephalon	5.69	3.68	2.85	5.23	4.36	1.32
whole brain	5.80	4.06	2.86	5.24	4.49	1.31

Table IV.3 Estimation of blood flows (ml/min) in each common carotid artery (CC), internal carotid artery and anastomoses between the external carotid and intracranial circulations. EC': blood flow in the external carotid artery beyond the bifurcation of the lingual artery.

Dog #	1	2	3	4	mean	SD
common carotid	162	115	173	109	140	32
EC'/CC	0.53	0.51	0.54	0.54	0.53	0.01
anastomoses	4.9	2.4	2.7	3.1	3.3	1.1
internal carotid	8.4	7.1	6.1	9.3	7.7	1.4

The first part of the document discusses the importance of maintaining accurate records of all transactions. This includes recording the date, amount, and purpose of each transaction. It also emphasizes the need for regular reconciliation of the accounts to ensure that the books are balanced and that there are no discrepancies.

The second part of the document provides a detailed explanation of the accounting cycle. It outlines the ten steps involved in the process, from identifying the accounting entities to preparing financial statements. Each step is described in detail, and examples are provided to illustrate the process.

The third part of the document discusses the various types of accounts used in accounting. It explains the difference between assets, liabilities, and equity accounts, and provides examples of each. It also discusses the importance of understanding the normal balances for each type of account.

The fourth part of the document provides a summary of the key concepts discussed in the previous sections. It emphasizes the importance of accuracy and integrity in accounting, and provides a final review of the accounting cycle and the types of accounts.

Account Name	Normal Balance	Debit	Credit
Assets	Debit	+	-
Liabilities	Credit	-	+
Equity	Credit	-	+
Income	Credit	-	+
Expenses	Debit	+	-
Dividends	Debit	+	-

Table IV.4 Plasma arginine vasopressin concentration (pg/ml) during control pre-infusion periods and during infusions of AII at 5.0 ng/kg/min into both common carotid arteries (CC), both external carotid arteries (EC), and the femoral vein (iv) for 10 min.

Dog #	Control	CC AII	Control	EC AII	Control	iv AII
1	1.7	4.2	1.0	1.0	3.0	3.4
2	0.9	3.8	0.8	1.5	1.7	2.5
3	0.8	1.5	1.2	1.6	0.5	1.4
4	1.8	3.3	1.6	3.2	1.7	1.8
5	2.4	2.7	2.6	3.9	3.3	2.5
6	2.5	3.6	2.8	2.9	3.4	2.7
7	4.1	7.4	4.7	6.4	5.6	5.8
mean	2.0	3.8	2.1	2.9	2.7	2.9
SD	1.1	1.8	1.4	1.8	1.6	1.4

Table IV.5 Plasma 11-hydroxycorticosteroids concentration ($\mu\text{g/ml}$) during control pre-infusion periods and during infusions of AII at 5.0 ng/kg/min into both common carotid arteries (CC), both external carotid arteries (EC), and the femoral vein (iv) for 10 min.

Dog #	Control	CC AII	Control	EC AII	Control	iv AII
1	1.7	6.3	2.3	8.0	3.3	2.7
2	3.6	6.8	3.4	5.5	2.7	2.0
3	2.8	4.4	2.2	3.5	2.0	1.4
4	1.1	2.9	1.8	3.9	2.8	2.2
5	1.9	4.1	2.0	3.4	2.2	1.9
mean	2.2	4.9	2.3	4.9	2.6	2.0
SD	1.0	1.6	0.6	2.0	0.5	0.5

Fig.IV.1 Schematic drawing of the carotid arteries and the anastomoses (A) between the external carotid and intracranial circulations. CC: common carotid artery. IC: internal carotid artery. EC': external carotid artery beyond the bifurcation of the lingual artery.

Fig. IV.1

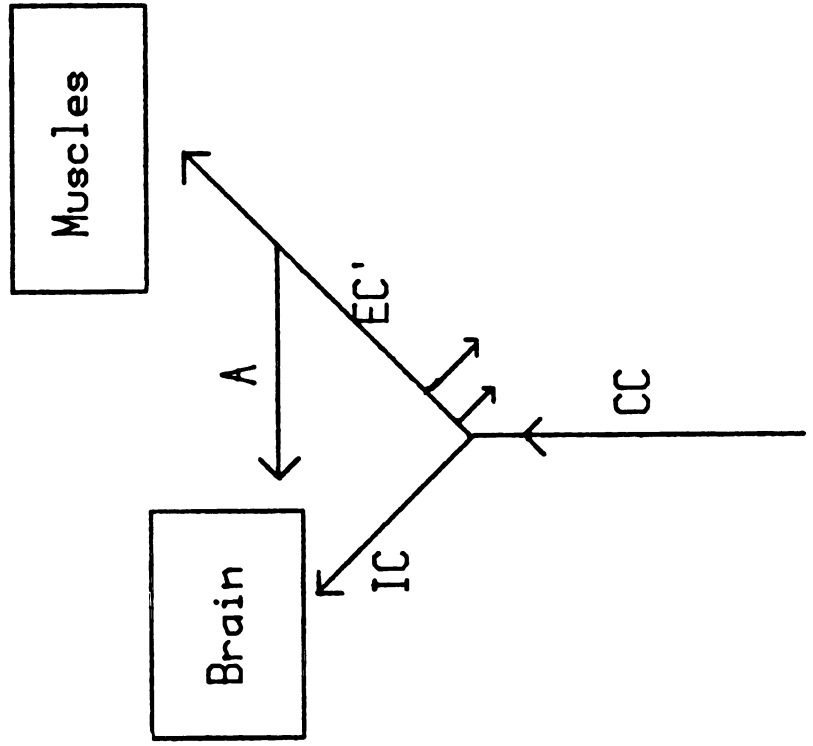


Fig.IV.2 Comparison of the distribution of microspheres in three parts of the telecephalon when injected into the common carotid artery (C.C.) vs that when injected into the external carotid artery (E.C.) in 3 dogs. The telecephalon was grossly divided into 3 parts: anterior, middle and posterior. The data are expressed as percentage of total spheres in the brain. Different symbols represent different dogs.

1. Деловые переговоры - это процесс, в котором участники обмениваются информацией и пытаются прийти к взаимовыгодному решению.

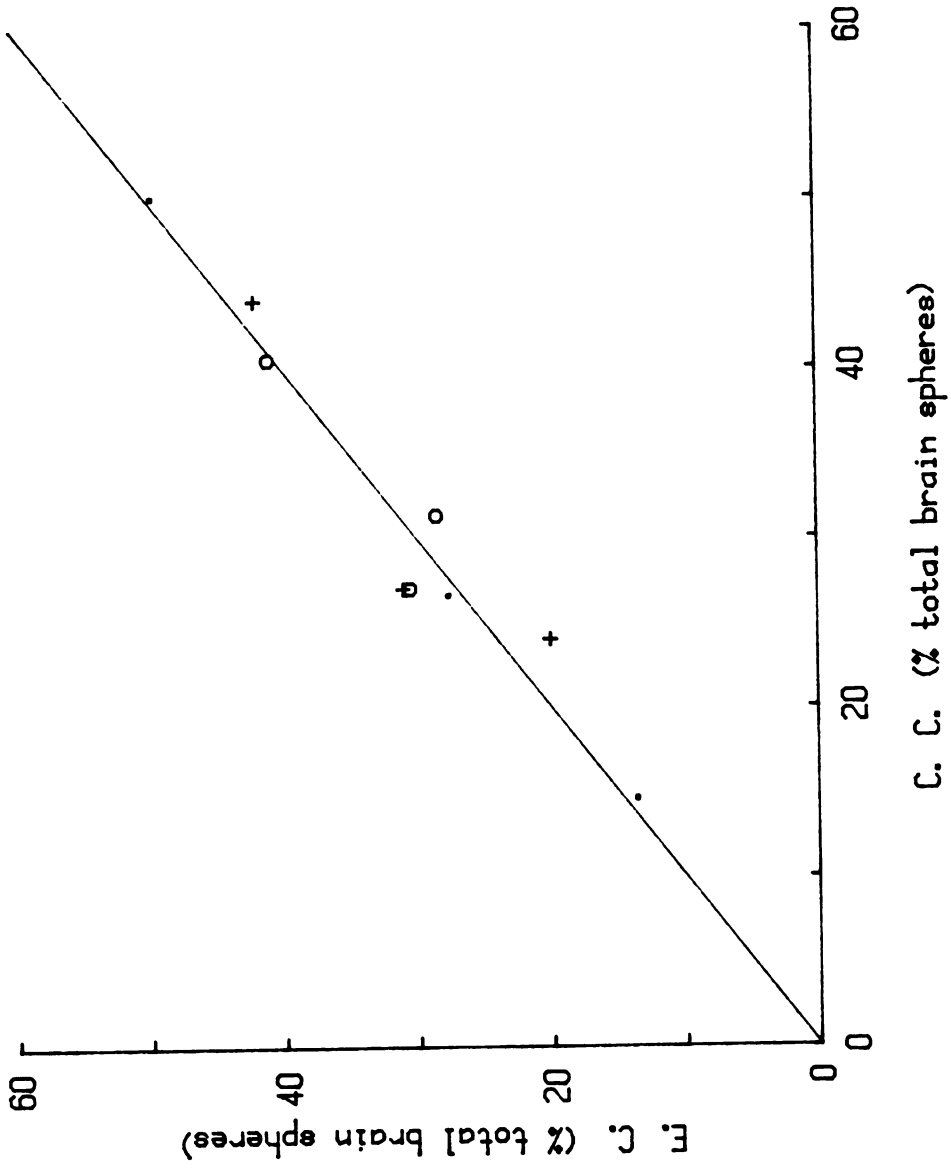
2. Цели переговоров могут быть различными: достижение соглашения, установление отношений, разрешение конфликта.

3. Участники переговоров могут быть индивидуальными или коллективными.

4. Процесс переговоров включает в себя несколько этапов: подготовка, открытие, предложение, торг, соглашение.

5. Результаты переговоров могут быть различными: соглашение, отказ, прекращение переговоров.

Fig. IV.2



CHAPTER V PRESSOR RESPONSES TO INTRACAROTID INFUSIONS
OF PHENYLEPHRINE AND VASOPRESSIN

As discussed earlier, the possibility exists that the pressor response to low doses of intracarotid AII is due to its extracranial vasoconstrictor effect. If this is true, intracarotid infusion of any other agents which cause extracranial vasoconstriction should also increase blood pressure more than intravenous infusion. To further test this hypothesis, the pressor responses to intracarotid infusions of two well-known vasoconstrictors, phenylephrine and vasopressin, were studied.

Phenylephrine, an α_1 -adrenergic agonist, was infused into the common carotid arteries on both sides or femoral vein at a rate of 1.0 $\mu\text{g}/\text{kg}/\text{min}$ for 10 min in 5 conscious dogs (19-22 kg). Synthetic arginine vasopressin (Bachem) was infused at a rate of 2.0 $\text{ng}/\text{kg}/\text{min}$ in 3 dogs (20-22 kg).

RESULTS

Intracarotid phenylephrine slightly increased mean arterial pressure from 95 ± 4 to 99 ± 7 mmHg (mean \pm standard deviation), whereas intravenous phenylephrine increased blood pressure from 95 ± 4 to 110 ± 8 mmHg (Fig.

The first part of the document discusses the importance of maintaining accurate records of all transactions. It emphasizes that every entry should be clearly documented, including the date, amount, and purpose of the transaction. This ensures transparency and allows for easy reconciliation of accounts.

In the second section, the author outlines the various methods used to collect and analyze data. These methods include direct observation, interviews, and the use of specialized software tools. Each method is described in detail, highlighting its strengths and limitations.

The third part of the document focuses on the results of the data analysis. It presents a series of tables and graphs that illustrate the trends and patterns observed in the data. The author provides a thorough interpretation of these results, explaining their significance and implications for the study.

Finally, the document concludes with a summary of the findings and a discussion of the study's limitations. The author suggests several areas for future research and provides recommendations for further investigation.

The following table provides a summary of the key data points discussed in the report.

Category	Value
Item A	12.5%
Item B	8.3%
Item C	15.7%
Item D	9.1%
Item E	11.4%

These results indicate a clear trend in the data, with Item C showing the highest percentage and Item B showing the lowest. Further analysis is required to understand the underlying causes of these trends.

V.1). The pressor response induced by intravenous phenylephrine was significantly greater than that induced by intracarotid phenylephrine ($p < 0.05$, paired t-test). Intracarotid phenylephrine seemed to decrease heart rate more than intravenous phenylephrine did in 4 dogs (-15 ± 5 vs -7 ± 6 beats/min, Fig. V.2). Statistical analysis was not done because of the small sample size.

Intracarotid vasopressin did not consistently alter the mean arterial pressure (mean change -2 ± 5 mmHg, Fig. V.3), whereas intravenous vasopressin increased blood pressure in all 3 dogs (mean increase 6 ± 1 mmHg). Both routes decreased heart rates in all 3 dogs (-9 ± 2 and -12 ± 7 beats/min, Fig. V.4).

DISCUSSION

If the vasoconstriction hypothesis is true, i.e., if extracranial vasoconstriction is responsible for the pressor response to intracarotid AII, intracarotid infusion of any other vasoconstrictor should increase blood pressure too. Both phenylephrine and vasopressin are potent vasoconstrictors. In sharp contrast to AII, however, intracarotid phenylephrine and vasopressin increased blood pressure to a less extent than intravenous infusions did (Figs. V.1 & 3). One possible explanation is that the vasoconstriction hypothesis

is not true and that both phenylephrine and vasopressin are cleared significantly through the cranial circulation. Therefore, smaller amounts of the vasoconstrictors reach general circulation and result in a smaller pressor response when they are infused into the carotid artery than when infused intravenously. However, since vasopressin is inactivated largely in the liver and kidneys and phenylephrine is metabolized mainly in the liver (Goodman & Gilman 1980), they are unlikely to be cleared significantly through the cranial circulation. Hence this explanation is probably not true.

Another possibility is that intracarotid phenylephrine and vasopressin could act on the brain to cause a depressor response. Although intraventricular α -agonists have been shown to lower blood pressure (Kaneko et al. 1960), systemically-administered phenylephrine has minimal central action (Goodman & Gilman 1980). Injection of norepinephrine and epinephrine into the internal carotid artery was reported not to decrease blood pressure in man (Greenfield & Tindall 1968). Furthermore, vasopressin has never been reported to have a central depressor action. Therefore, this possibility is unlikely to be true.

Finally, intracarotid phenylephrine and vasopressin could act on the carotid sinus to increase

baroreceptor activity and hence cause a depressor effect. Intracarotid infusions delivered a much higher concentration of drugs to the carotid sinus than intravenous infusions did. Heymans & Delaunois (1951) showed that constriction of the carotid sinus wall was responsible for the increased baroreceptor stimulation leading to hypotension and diminution of the carotid occlusion reflex when epinephrine or norepinephrine was applied locally. Vasopressin and other vasoconstrictors other than AII have also been shown to have the similar effect (Heymans 1955). This view is supported by the present finding that even though it caused a smaller pressor response, intracarotid phenylephrine, presumably acting on the carotid sinus, induced a greater fall in heart rate than intravenous infusion did (Fig. V.2). On the other hand, McCubbin et al. (1957) showed that AII had no effect on the carotid sinus. This point is supported by the finding presented in Chapter III that external carotid infusion of AII increased blood pressure to the same degree as common carotid infusion did and that both routes of infusion did not alter heart rate significantly. By recording the action potentials in single baroreceptor fibers, Lumbers et al. (1979) also concluded that AII had no effect on the sensitivity of carotid sinus baroreceptor. Therefore, it is possible that the

pressor effects of intracarotid phenylephrine and vasopressin caused by extracranial vasoconstriction, unlike AII, might be masked by their depressor effects induced by their direct action on the carotid sinus.

In conclusion, due to the possible direct actions of phenylephrine and vasopressin on the carotid sinus, this study does not rule out the possibility that extracranial vasoconstriction is responsible for the pressor response to intracarotid AII.

Fig.V.1 The pressor response to intravenous (iv) phenylephrine (PhE) was greater than that to common carotid infusion (CC) in 5 conscious dogs. Phenylephrine was infused at 1.0 $\mu\text{g}/\text{kg}/\text{min}$ for 10 min. Different symbols represent different dogs. Control: pre-infusion control period.

- 1990年10月，在《人民日报》发表《中国要实行股份制》一文，提出“股份制是公有制的一种实现形式”
- 1992年10月，在党的十四大上，提出“公有制实现形式可以而且应当多样化，一切反映社会化生产规律的经营方式和组织形式都可以大胆利用，要努力寻找能够极大促进生产力发展的公有制实现形式”
- 1997年9月，在党的十五大上，提出“公有制实现形式可以而且应当多样化，一切反映社会化生产规律的经营方式和组织形式都可以大胆利用，要努力寻找能够极大促进生产力发展的公有制实现形式”
- 1998年12月，在党的十五届三中全会通过的《中共中央关于农业、农村、农民若干重大问题的决定》中，提出“要积极探索公有制实现形式多样化的有效途径”

Fig. V.1

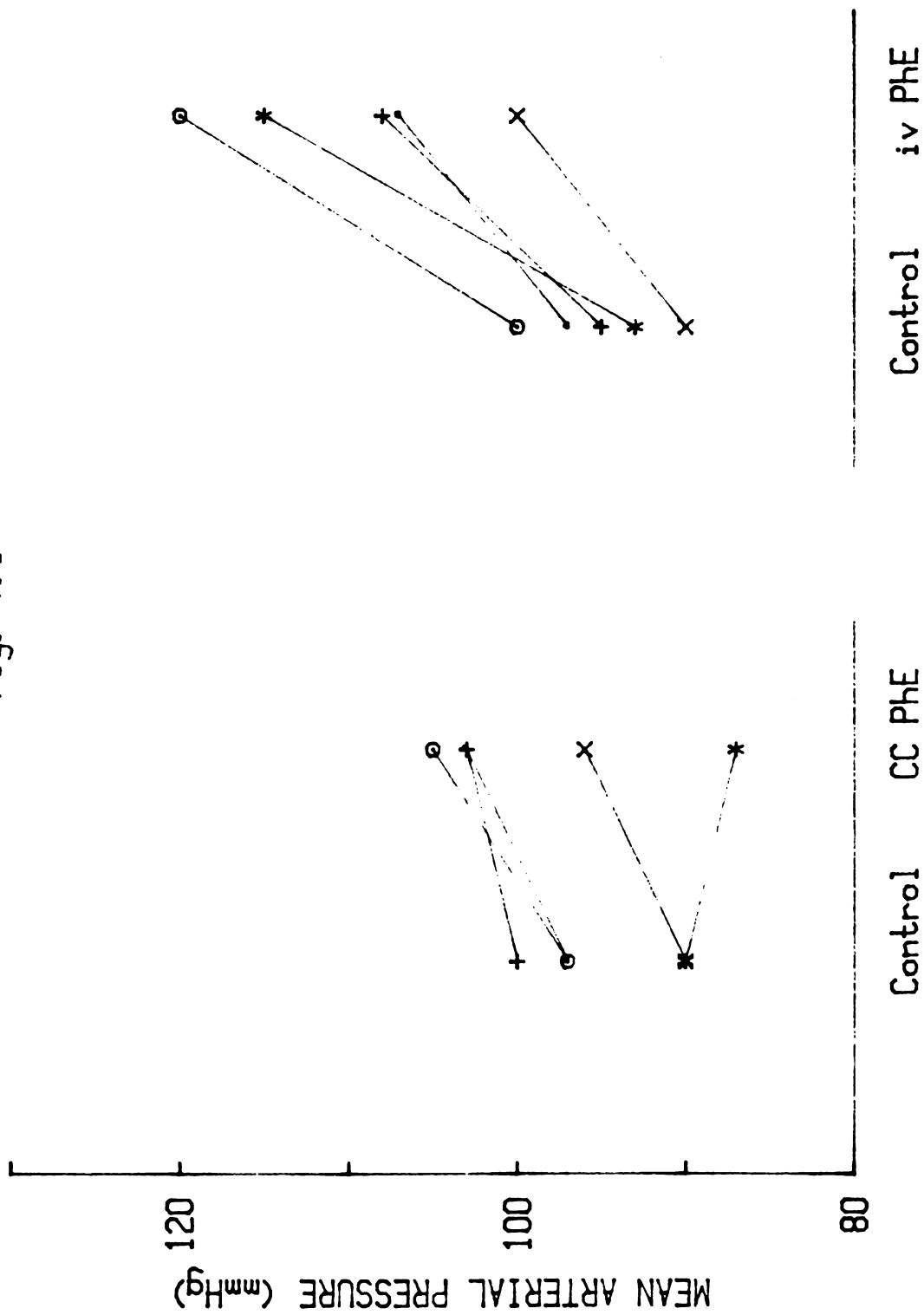


Fig.V.2 Mean heart rate during control pre-infusion periods and during intracarotid and intravenous phenylephrine in 4 dogs. Intracarotid phenylephrine appeared to decrease heart rate more than i.v. infusion.

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Fig. V.2

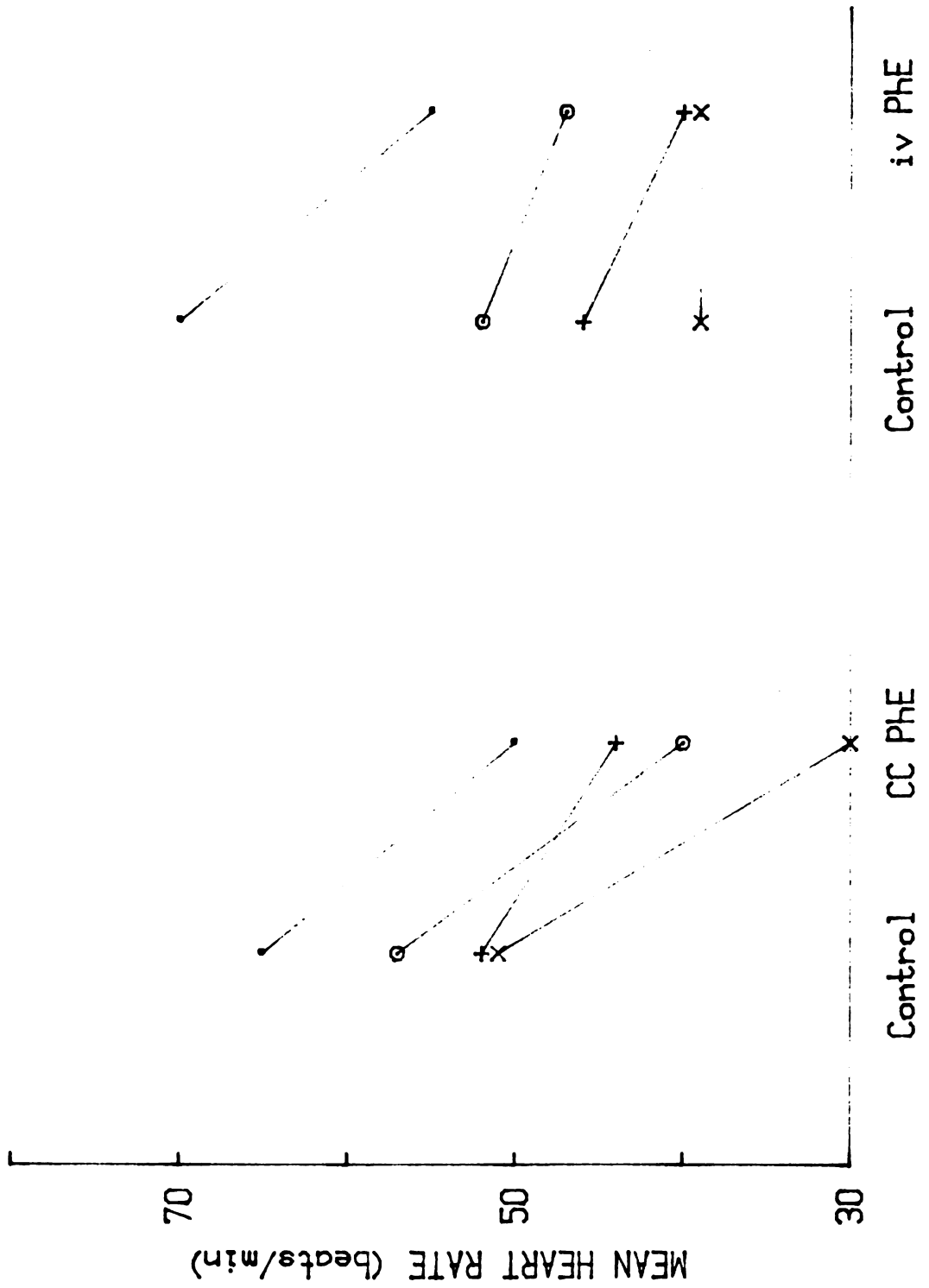


Fig.V.3 Mean arterial pressure during control pre-infusion periods and during intracarotid and intravenous infusion of arginine vasopressin (AVP) in 3 dogs. Vasopressin was infused at 2.0 ng/kg/min for 10 min.

1982年12月25日，在党的十一届三中全会召开三周年之际，邓小平同志在中央工作会议上，第一次提出了“建设有中国特色的社会主义”这个命题。1987年10月，党的十三大正式提出“建设有中国特色的社会主义”这个命题。1989年6月，党的十三届四中全会进一步提出“建设有中国特色的社会主义”这个命题。1992年10月，党的十四大正式提出“建设有中国特色的社会主义”这个命题。1997年9月，党的十五大正式提出“建设有中国特色的社会主义”这个命题。1999年9月，党的十五届四中全会进一步提出“建设有中国特色的社会主义”这个命题。2002年11月，党的十六大正式提出“建设有中国特色的社会主义”这个命题。2007年10月，党的十七大正式提出“建设有中国特色的社会主义”这个命题。2012年11月，党的十八大正式提出“建设有中国特色的社会主义”这个命题。2017年10月，党的十九大正式提出“建设有中国特色的社会主义”这个命题。2022年10月，党的二十大正式提出“建设有中国特色的社会主义”这个命题。

Fig. V.3

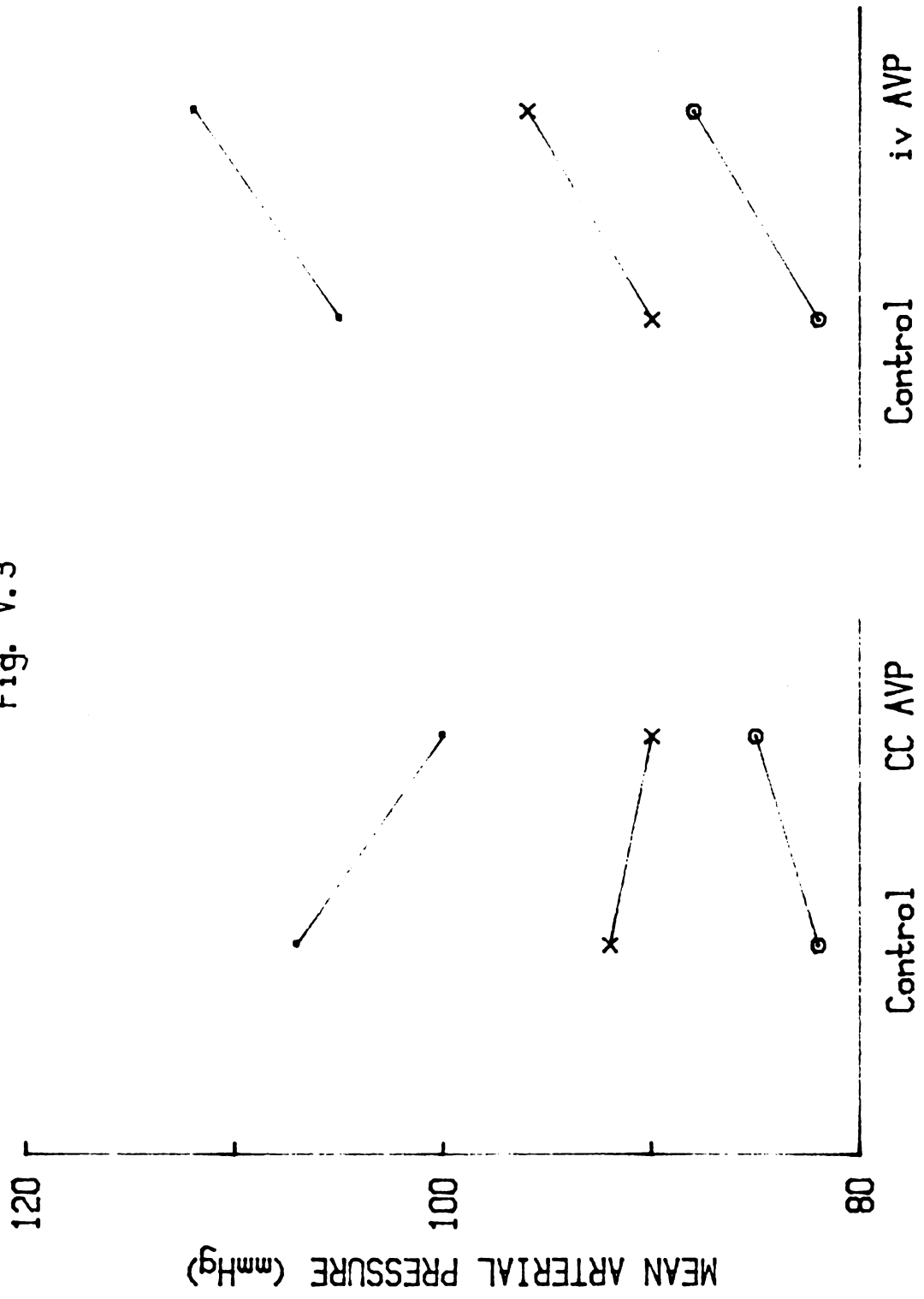
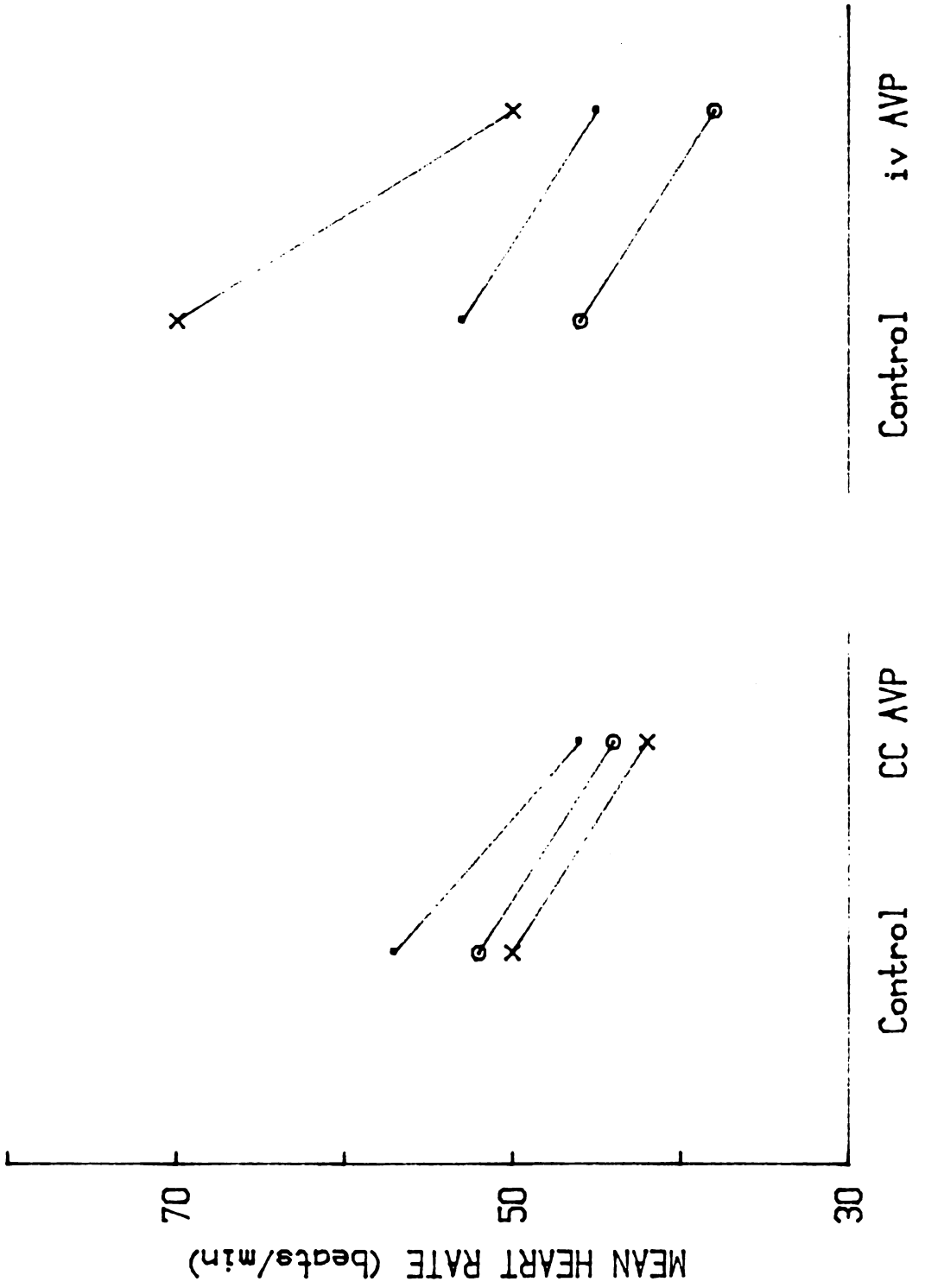


Fig.V.4 Mean heart rate during control pre-infusion periods and during intracarotid and intravenous infusion of arginine vasopressin in 3 dogs.

- $\frac{1}{2} \int_{-\infty}^{\infty} \delta(x) dx = \frac{1}{2}$
- $\int_{-\infty}^{\infty} \delta(x) dx = 1$
- $\int_{-\infty}^{\infty} \delta(x) f(x) dx = f(0)$

Fig. V.4



CHAPTER VI EFFECTS OF INTRAVENTRICULAR SARALASIN ON
THE PRESSOR RESPONSE TO BLOOD-BORNE ANGIOTENSIN II

INTRODUCTION

As discussed previously, administration of AII into the vertebral arteries has been demonstrated to elicit a centrally mediated pressor response in dogs (Lowe & Scroop 1969, Ferrario et al. 1970), rabbits (Yu & Dickinson 1965) and man (Ueda et al. 1969). Blood-borne AII can therefore act not only on the vascular smooth muscles but also on the brain to increase arterial blood pressure. However, to what extent the central pressor component contributes to the pressor response to blood-borne AII is still unsettled.

Ablation of the area postrema in the greyhound not only abolished the pressor action of intravertebral AII (Joy & Lowe 1970b) but also reduced that of intravenous AII (Scroop et al. 1971). This lesion also lowered blood pressure following hemorrhage (Katic et al. 1971) and impaired the development of acute renal hypertension (Scroop et al. 1975). Hence, the central pressor component of circulating AII appeared to be important in greyhounds.

However, the area postrema is very close to the

nucleus tractus solitarii, an important medullary site of blood pressure regulation (Crill & Reis 1968). Ablation of the area postrema might damage this adjacent nucleus as well. Furthermore, these studies were done on anesthetized greyhounds. As the complications of anesthesia are well-known, there seems to be a need to examine this question in conscious dogs by using different approach.

In the present study, saralasin, a competitive antagonist of AII, was infused into the third cerebroventricle of conscious dogs. Its effects on the pressor responses to intravertebral, intracarotid and intravenous AII were then compared. The pressor response to intravenous AII was to mimic the overall pressor effect of circulating AII, and that to intravertebral and intracarotid AII to mimic its central pressor component.

METHODS

A cannula was chronically implanted in the third cerebroventricle of 5 mongrel dogs (20-25 kg). The cannula was a 20-gauge stainless steel tubing (Small Parts, Miami) bent at a 120°-angle in one end. The cannula was placed stereotaxically through a hole drilled in the skull and was held in place by dental

cement with 4 stainless steel screws fixed in the skull. The placement of the cannula was determined by X-ray photography after injecting 0.3 ml meglumine iothalamate (Conray) through the cannula (Thrasher et al. 1980). A tygon tubing was connected to the end of the cannula, passed subcutaneously and exteriorized at the back of the dog.

One week later, the second surgery was performed. Small, non-occluding catheters were implanted in vertebral and common carotid arteries on both sides for AII infusion. Two other catheters were placed in a femoral artery and vein for blood pressure recording and AII infusion, respectively.

The dogs were allowed to recover from the surgery for another week. On the day of the experiment, the dog was brought into the laboratory. Saralasin or artificial cerebrospinal fluid (CSF) was infused into the third ventricle for 60 min, AII was then infused into both carotid arteries, both vertebral arteries or femoral vein in random order. Arterial blood pressure and heart rate were monitored throughout the experiment. Consecutive experiments were separated by at least one day apart to avoid any carry-over effects.

Saralasin ([Sar¹-Ile⁵-Ala⁸] angiotensin II,

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it more precisely. This involves determining the scope of the problem, its causes, and its effects. A clear definition of the problem is essential for developing an effective solution.

2. The second step is to analyze the problem. This involves gathering information about the problem and its context. This can be done through interviews, surveys, or other data collection methods. The goal is to understand the underlying causes of the problem and to identify any constraints or limitations that may affect the solution. Once the problem has been analyzed, the next step is to generate potential solutions.

3. The third step is to generate potential solutions. This involves brainstorming ideas and evaluating them based on their feasibility, effectiveness, and cost. It is important to consider a wide range of options and to evaluate them based on objective criteria. Once a potential solution has been identified, the next step is to develop a plan for implementing it.

4. The fourth step is to develop a plan for implementing the solution. This involves determining the resources needed, the timeline, and the responsibilities of the individuals involved. A clear plan is essential for ensuring that the solution is implemented effectively. Once the plan has been developed, the next step is to implement the solution.

5. The fifth step is to implement the solution. This involves putting the plan into action and monitoring progress. It is important to communicate the plan to all relevant parties and to ensure that they understand their roles and responsibilities. Once the solution has been implemented, the final step is to evaluate the results.

6. The sixth step is to evaluate the results. This involves comparing the actual performance with the desired state and determining whether the solution has been effective. If the solution has not been effective, it may be necessary to re-evaluate the problem and generate new solutions. If the solution has been effective, it is important to document the results and to share them with others who may be facing similar problems.

Bachem) was freshly prepared in artificial CSF. The composition of the artificial CSF was described by Thrasher et al. (1980). Its pH was approximately 7.4 and was buffered by bicarbonate and phosphate. Saralasin was infused at a rate of 0.1 $\mu\text{g}/\text{kg}/\text{min}$ (in 5 $\mu\text{l}/\text{min}$). Angiotensin II was infused at a rate of 1.0 $\text{ng}/\text{kg}/\text{min}$ into a carotid artery, 0.5 $\text{ng}/\text{kg}/\text{min}$ into a vertebral artery, or 2, 5 and 10 $\text{ng}/\text{kg}/\text{min}$ into a femoral vein.

Student's paired t-test was used to analyze the effects of saralasin on the blood pressure and heart rate changes during AII infusions into the vertebral or carotid artery. Analysis of variance for repeated measurements was used to analyze those during intravenous infusion. Data are expressed as mean \pm standard deviation.

RESULTS

The control mean arterial pressure and heart rate during intraventricular infusion of artificial CSF were 115 \pm 9 mmHg and 82 \pm 16 beats/min, respectively. They were 115 \pm 12 mmHg and 90 \pm 8 beats/min, respectively, during intraventricular saralasin infusion. When AII was infused into the vertebral arteries, the mean blood pressure and heart rate increased promptly by 15 \pm 6 mmHg

and 19 ± 12 beats/min, respectively, during intraventricular artificial CSF infusion (Fig.VI.1, 2 & 3). Intraventricular saralasin significantly inhibited these increases; the mean changes in blood pressure and heart rate were 4 ± 6 mmHg and -2 ± 5 beats/min, respectively, to intravertebral AII (Fig.VI.1, 2 & 3).

On the other hand, intraventricular saralasin had no effect on the pressor response to intracarotid AII (10 ± 5 vs 10 ± 4 mmHg, Fig.VI.2). Saralasin also had no effect on the pressor responses to intravenous AII at three different doses (Fig.VI.4, Table VI.1). Heart rate did not change significantly to intracarotid (Fig.VI.3) and intravenous AII (Table VI.2) during intraventricular infusion of either artificial CSF or saralasin.

DISCUSSION

When AII is infused intravenously, it acts peripherally to cause systemic vasoconstriction; however, it also has access to the brain where it can exert a central pressor action. Since intraventricular saralasin inhibited the pressor action of intravertebral AII (Fig.VI.2), one would expect that saralasin would also reduce the pressor response to AII i.v. if the central pressor action of intravertebral AII was

— $\frac{1}{2} \times 100 = 50\%$ (the probability of a child being born with a normal genotype is 50%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a normal phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a recessive phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a dominant phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a dominant genotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a recessive genotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a normal genotype and a normal phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a normal genotype and a recessive phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a dominant genotype and a normal phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a dominant genotype and a recessive phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a recessive genotype and a normal phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a recessive genotype and a recessive phenotype is 25%)

— $\frac{1}{4} \times 100 = 25\%$ (the probability of a child being born with a normal genotype and a normal phenotype is 25%)

important. However, this was not so (Fig.VI.4). It is therefore concluded that the central pressor action of intravertebral AII is not essential in the pressor response to blood-borne AII.

Another finding in keeping with this conclusion is the heart rate response. Intraventricular saralasin inhibited the tachycardia produced by intravertebral AII (Fig.VI.3). This central action of intravertebral AII appeared not to be essential in the maintenance of heart rate during i.v. AII, because saralasin did not lower heart rate when AII was given intravenously (Table VI.2).

The present conclusion, however, is contrary to previous reports on greyhounds. The central pressor action of intravertebral AII was shown to be important in the pressor response to AII i.v. (Scroop et al. 1971), in the maintenance of blood pressure following hemorrhage (Katic et al. 1971) and in the development of acute renal hypertension (Scroop et al. 1975).

It is difficult to explain this discrepancy. However, species difference is one possible explanation. Mongrel dogs and greyhounds are different in that the pressor response to intravertebral AII is mediated mainly by withdrawal of the vagal tone to the heart in

greyhounds (Scroop & Lowe 1969), whereas by increased efferent sympathetic activity in mongrel dogs (Ferrario et al. 1972). In addition, studies on the greyhounds were all done during morphine-chloralose anesthesia, whereas the present study was carried out in conscious dogs. In addition to the effects of anesthesia, the trauma caused by extensive surgery can only be avoided by working on conscious animals fully recovered from surgery.

Furthermore, ablation of the area postrema might also damage adjacent nuclei such as the nucleus tractus solitarii, the site of the first medullary relay in the baroreceptor reflex arc (Crill & Reis 1968). In some of the studies on greyhounds, either the control heart rate (Scroop et al. 1971) or blood pressure (Scroop et al. 1975) appeared to be elevated after the lesion. This could be due to the unexpected damage of this adjacent nucleus (Zandberg et al. 1977).

On the other hand, the present finding is in agreement with some other studies. Ramsay et al. (1978) reported that intraventricular saralasin had no effect on the pressor response to AII i.v. in pentobarbital-anesthetized mongrel dogs. Reid (1976) found that intraventricular saralasin had no effect on the maintenance of blood pressure in sodium-depleted

the first of these is the fact that the system is not a simple one. It is a complex system with many interacting components. The second is that the system is not a closed system. It is an open system that interacts with its environment. The third is that the system is not a linear system. It is a non-linear system with many feedback loops. The fourth is that the system is not a static system. It is a dynamic system that changes over time. The fifth is that the system is not a deterministic system. It is a stochastic system with many random elements. The sixth is that the system is not a simple system. It is a complex system with many interacting components. The seventh is that the system is not a closed system. It is an open system that interacts with its environment. The eighth is that the system is not a linear system. It is a non-linear system with many feedback loops. The ninth is that the system is not a static system. It is a dynamic system that changes over time. The tenth is that the system is not a deterministic system. It is a stochastic system with many random elements.

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mongrel dogs, whose plasma AII concentrations were elevated. Most recently, by comparing the effects of saralasin infusions into the vertebral artery, carotid artery and femoral vein on the blood pressure, Brooks & Reid (1981) also concluded that the effect of AII on the maintenance of blood pressure in sodium-depleted dogs was not mediated via the brain. In the rat, intraventricular saralasin had no effect on the blood pressure following ligation of the abdominal vena cava (to be presented in Chapter VII), a procedure that increased the plasma AII concentration.

Sweet et al. (1977) showed that intraventricular administration of the antagonist, [Sar¹-Ile⁸] AII, did not affect the pressor response to AII i.v. in rats, which is in keeping with the present study. Although the same treatment lowered blood pressure in experimental renal hypertensive rats, this finding did not lead to the conclusion that the central pressor component was important. The reason is that the antagonist also lowered blood pressure in the spontaneously hypertensive rats, whose plasma AII concentrations were not elevated (Mann et al. 1978); thus this treatment did not specifically inhibit the pressor action of circulating AII. Similar results were obtained by using saralasin (Mann et al. 1978).

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance against a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define the problem more precisely. This involves determining the scope of the problem, the time frame, and the specific areas affected. For instance, a manager might determine that the problem is limited to a specific product line or geographic region. The third step is to analyze the causes of the problem. This is often done by using tools such as the fishbone diagram (Ishikawa diagram) to identify the root causes of the problem. The fourth step is to develop a plan of action to address the problem. This involves identifying the specific actions that need to be taken, the resources required, and the timeline for implementation. The fifth and final step is to implement the plan and monitor the results. This involves tracking the progress of the plan and making adjustments as needed to ensure that the problem is resolved.

Lesions of the anteroventral third ventricle have been reported to depress the pressor response to AII i.v., and to lower blood pressure in renal hypertensive rats (Buggy et al. 1977). However, the same lesion also lowered blood pressure in several forms of low-renin hypertension, such as deoxycorticosterone-salt and spontaneous hypertension (Brody et al. 1978). Therefore, this lesion did not specifically inhibit the pressor action of circulating AII, and these observations did not provide further information on the significance of the central pressor component of circulating AII.

There was a recent report that subfornical organ lesion reduced the pressor effect of i.v. AII in rats (Mangiapane & Simpson 1980). However, since this lesion also inhibited the dipsogenic effect of i.v. AII (Simpson et al. 1978), the possibility exists that the central pressor component inhibited by the lesion is due to the arousal effect of AII. More research is needed to clarify the specificity of this lesion.

Since the pressor response to intravertebral, but not intracarotid, AII was inhibited by saralasin (Fig.VI.2), these results thus provide direct evidence that the mechanism of the pressor response to intracarotid AII is different from that to intravertebral

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it more precisely. This involves determining the scope of the problem, its causes, and its potential consequences.

2. The second step is to analyze the problem. This involves gathering information about the problem and its context. This can be done through various methods, such as interviews, surveys, and data analysis. The goal is to understand the underlying causes of the problem and to identify any constraints or limitations that may affect the solution. For example, a manager might analyze sales data to identify trends and patterns, or they might interview customers to understand their needs and expectations.

3. The third step is to generate potential solutions. This involves brainstorming ideas and evaluating them based on their feasibility and effectiveness. This can be done through various methods, such as group brainstorming, individual brainstorming, and decision-making techniques. The goal is to identify a solution that addresses the problem and is consistent with the organization's goals and values. For example, a manager might generate several potential solutions for declining sales, such as increasing marketing efforts, improving customer service, or offering discounts.

4. The fourth step is to implement the chosen solution. This involves putting the solution into action and monitoring its progress. This can be done through various methods, such as developing a plan, assigning responsibilities, and tracking performance. The goal is to ensure that the solution is implemented effectively and that the problem is resolved. For example, a manager might implement a new marketing campaign and track sales performance over time to see if it leads to an increase in sales.

5. The fifth and final step is to evaluate the results. This involves assessing the effectiveness of the solution and identifying any areas for improvement. This can be done through various methods, such as comparing current performance with the desired state, conducting surveys, and analyzing data. The goal is to ensure that the solution has been implemented successfully and that the problem has been resolved. For example, a manager might evaluate the results of a new marketing campaign by comparing sales performance before and after the campaign.

6. The sixth step is to communicate the results. This involves sharing the findings of the problem-solving process with relevant stakeholders. This can be done through various methods, such as reports, presentations, and meetings. The goal is to ensure that all stakeholders are aware of the problem, the solution, and the results. For example, a manager might present the results of a problem-solving process to a team meeting or a board of directors.

7. The seventh step is to learn from the experience. This involves reflecting on the problem-solving process and identifying lessons learned. This can be done through various methods, such as debriefing, reflection, and documentation. The goal is to improve the organization's ability to identify and solve problems in the future. For example, a manager might debrief a team after a problem-solving process to identify what worked well and what could be improved.

8. The eighth step is to document the process. This involves creating a record of the problem-solving process, including the problem, the analysis, the solutions, and the results. This can be done through various methods, such as reports, presentations, and meetings. The goal is to ensure that the process is documented and can be used as a reference for future problem-solving efforts. For example, a manager might create a report documenting the problem-solving process for a declining sales problem.

AII. This is in keeping with the result of the microsphere study presented in Chapter IV, where carotid blood was shown not to perfuse the medulla. So the site of action of intracarotid AII must not lie in the medulla where the intravertebral AII acts to raise blood pressure. In addition, intraventricular saralasin also inhibited drinking and vasopressin secretion to AII i.v. (Thrasher & Ramsay, unpublished observation), hence the sites of these AII actions must be different from that of the pressor action of intracarotid AII.

In the rat, intracarotid rather than intravertebral AII was recently reported to cause a greater pressor response than intra-aortic AII did (Haywood et al. 1980). The pressor response to intracarotid AII was inhibited by intraventricular saralasin. Rats thus appear to be different from dogs in the central pressor actions of circulating AII. The subfornical organ (Mangiapane & Simpson 1980) and the organum vasculosum of the lamina terminalis (Fink et al. 1980) have been implicated to contain receptors for the pressor effect of AII in rats.

In conclusion, the central pressor action of intravertebral AII appears to be not essential in the pressor response to blood-borne AII in conscious

1. The first part of the text discusses the importance of maintaining accurate records in a business context. It highlights how proper record-keeping can help in identifying trends, resolving disputes, and ensuring compliance with legal requirements. The author emphasizes that records should be kept for a sufficient period to allow for a thorough review if needed.

2. The second part of the text focuses on the challenges of data management in the digital age. It notes that while digital records are convenient, they also pose risks such as data loss, security breaches, and information overload. The author suggests implementing robust backup systems and access controls to mitigate these risks.

3. The third part of the text addresses the issue of data privacy. It discusses the need to protect sensitive information from unauthorized access and the importance of obtaining consent from individuals whose data is being collected. The author mentions various regulations, such as the GDPR, that govern data privacy practices.

4. The final part of the text concludes by reiterating the significance of data management for business success. It encourages organizations to adopt a proactive approach to data management, ensuring that their records are accurate, secure, and accessible when needed.

5. The first part of the text discusses the importance of maintaining accurate records in a business context. It highlights how proper record-keeping can help in identifying trends, resolving disputes, and ensuring compliance with legal requirements. The author emphasizes that records should be kept for a sufficient period to allow for a thorough review if needed.

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mongrel dogs. In addition, the sites of the pressor actions of intracarotid and intravertebral AII are different.

Table VI.1 Lack of effect of intraventricular saralasin on the mean arterial blood pressure (mmHg) during i.v. AII at 2.0, 5.0 and 10 ng/kg/min for 10 min. (a) control intraventricular artificial CSF infusion, (b) intraventricular saralasin infusion (0.1 μ g/kg/min).

1. The first step in the process of identifying a problem is to define the problem. This involves identifying the symptoms and the underlying causes of the problem. Once the problem has been defined, the next step is to identify the stakeholders who are affected by the problem. This includes identifying the individuals, groups, and organizations that are impacted by the problem. The third step is to gather information about the problem. This involves conducting research and collecting data that will help to understand the problem more fully. The fourth step is to analyze the information that has been gathered. This involves identifying the key issues and the potential solutions to the problem. The fifth step is to develop a plan of action. This involves identifying the specific steps that need to be taken to address the problem. The sixth step is to implement the plan. This involves putting the plan into action and monitoring the progress. The seventh step is to evaluate the results. This involves assessing the effectiveness of the plan and making adjustments as needed. The eighth step is to communicate the results. This involves sharing the findings with the stakeholders and the public. The ninth step is to document the process. This involves creating a record of the steps that were taken and the results that were achieved. The tenth step is to review the process. This involves reflecting on the experience and identifying lessons learned for future reference.

(a) Artificial CSF

Dog #	Control	AII (2.0)	Control	AII (5.0)	Control	AII (10)
1	97	103	95	120	100	140
2	95	100	107	117	100	117
3	115	123	113	127	110	130
4	105	110	110	120	108	123
5	103	108	102	115	105	120
mean	103	109	105	120	105	126
SD	8	9	7	5	5	9

(b) Saralasin

1	102	110	100	122	100	135
2	94	98	93	101	93	120
3	113	118	115	127	110	128
4	110	117	115	125	120	132
5	102	105	103	112	102	117
mean	104	110	105	117	105	126
SD	7	8	10	11	10	8

1. $\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$

2. $\frac{1}{2} \times \frac{1}{3} = \frac{1}{6}$

3. $\frac{1}{2} \times \frac{1}{4} = \frac{1}{8}$

4. $\frac{1}{2} \times \frac{1}{5} = \frac{1}{10}$

5. $\frac{1}{2} \times \frac{1}{6} = \frac{1}{12}$

6. $\frac{1}{2} \times \frac{1}{7} = \frac{1}{14}$

7. $\frac{1}{2} \times \frac{1}{8} = \frac{1}{16}$

8. $\frac{1}{2} \times \frac{1}{9} = \frac{1}{18}$

9. $\frac{1}{2} \times \frac{1}{10} = \frac{1}{20}$

10. $\frac{1}{2} \times \frac{1}{11} = \frac{1}{22}$

11. $\frac{1}{2} \times \frac{1}{12} = \frac{1}{24}$

Table VI.2 Lack of effect of intraventricular saralasin on the mean heart rate (beats/min) during i.v. AII at 2.0, 5.0 and 10 ng/kg/min for 10 min. (a) control intraventricular artificial CSF infusion, (b) intraventricular saralasin infusion (0.1 μ g/kg/min).

(a) Artificial CSF

Dog #	Control	AII (2.0)	Control	AII (5.0)	Control	AII (10)
1	70	60	60	55	0	85
2	60	50	57	45	62	48
3	55	40	50	50	45	35
4	65	61	61	66	62	64
5	83	83	83	77	80	80
mean	67	59	62	59	64	62
SD	11	16	12	13	13	21

(b) Saralasin

1	55	50	70	65	67	55
2	65	58	62	60	60	65
3	70	65	65	55	65	60
4	70	66	80	82	70	80
5	83	83	80	75	78	75
mean	69	64	71	67	68	67
SD	10	12	8	11	7	10

1. The first part of the document discusses the importance of maintaining accurate records of all transactions.

2. It also emphasizes the need for regular audits to ensure the integrity of the financial data.

3. Furthermore, the document highlights the role of transparency in building trust with stakeholders.

4. In addition, it outlines the various methods used to collect and analyze financial information.

5. The document also addresses the challenges associated with data security and privacy.

6. Moreover, it discusses the impact of technological advancements on financial reporting.

7. Finally, the document concludes by emphasizing the importance of continuous improvement in financial management practices.

8. The following table provides a summary of the key findings and recommendations.

9. It is important to note that these findings are based on a limited sample size and may not be representative of the entire population.

10. The data indicates that there is a significant correlation between the variables studied.

11. The results suggest that there is a need for further research in this area.

12. The findings also highlight the need for improved data collection methods.

13. The study concludes that there is a need for more comprehensive data analysis.

14. The results indicate that there is a need for more robust data security measures.

Fig.VI.1 A typical example of the inhibition effects of intraventricular saralasin on the pressor and tachycardia responses to intravertebral AII. Upper panel: intraventricular control infusion of artificial CSF (aCSF). Lower panel: intraventricular infusion of saralasin (0.1 $\mu\text{g}/\text{kg}/\text{min}$). MAP: mean arterial blood pressure (mmHg), HR: heart rate (beats/min). Angiotensin was infused at 0.5 $\text{ng}/\text{kg}/\text{min}$ into each artery between the arrows.

1. **Introduction** (10%)
 2. **Background** (10%)
 3. **Methodology** (10%)
 4. **Results** (10%)
 5. **Discussion** (10%)
 6. **Conclusion** (10%)
 7. **References** (10%)
 8. **Appendix** (10%)
 9. **Summary** (10%)
 10. **Final Remarks** (10%)

Fig. VI.1

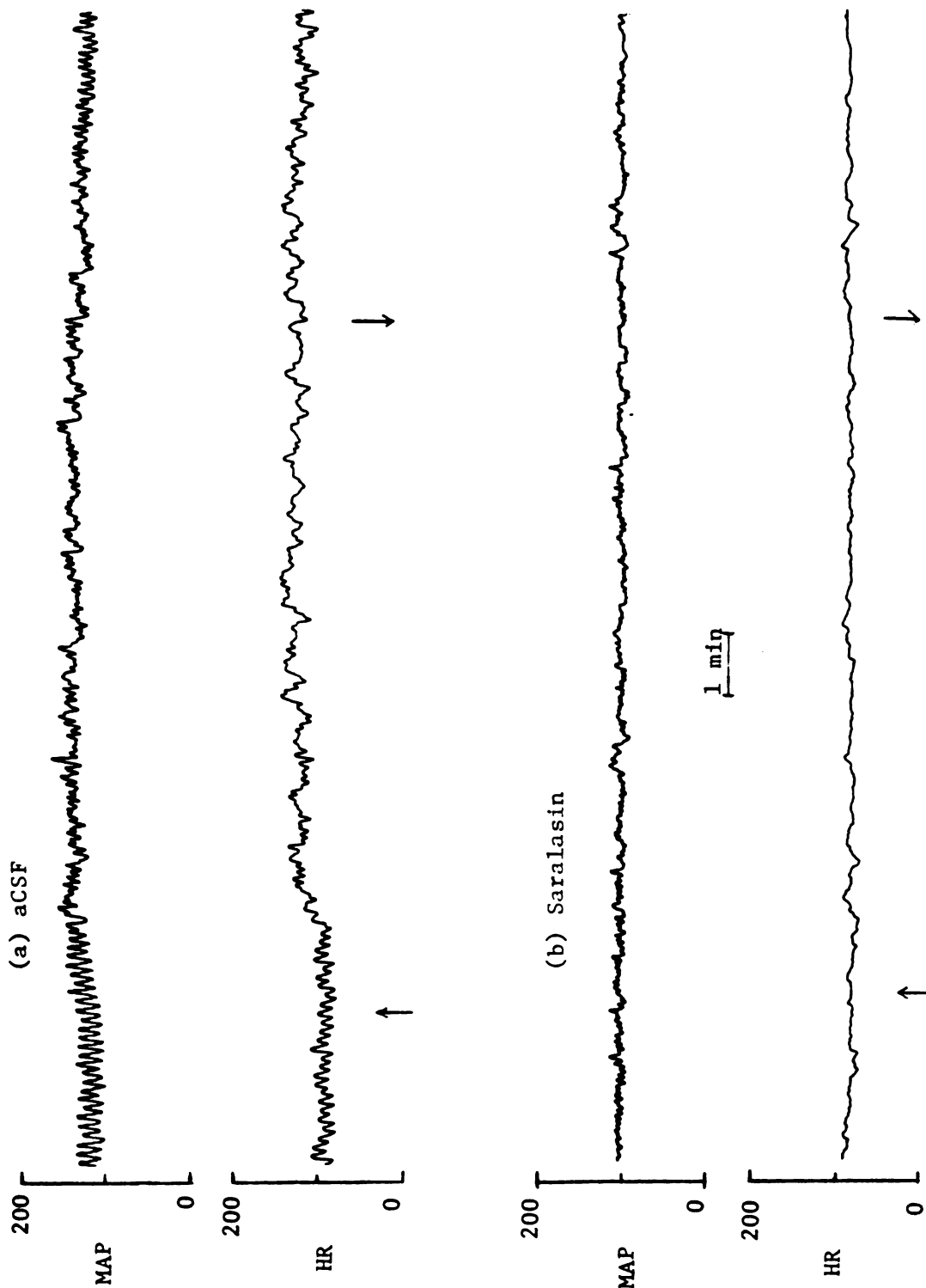


Fig.VI.2 Effects of intraventricular saralasin (Sar) on the pressor responses to intravertebral (left) and intracarotid AII in 5 dogs. Saralasin significantly reduced the pressor effect of intravertebral AII ($p < 0.05$). The AII infusion rates were 1.0 and 2.0 ng/kg/min for the vertebral and common carotid arteries, respectively. aCSF: intraventricular control infusion of artificial CSF. Different symbols represent different dogs.

Fig. VI.2

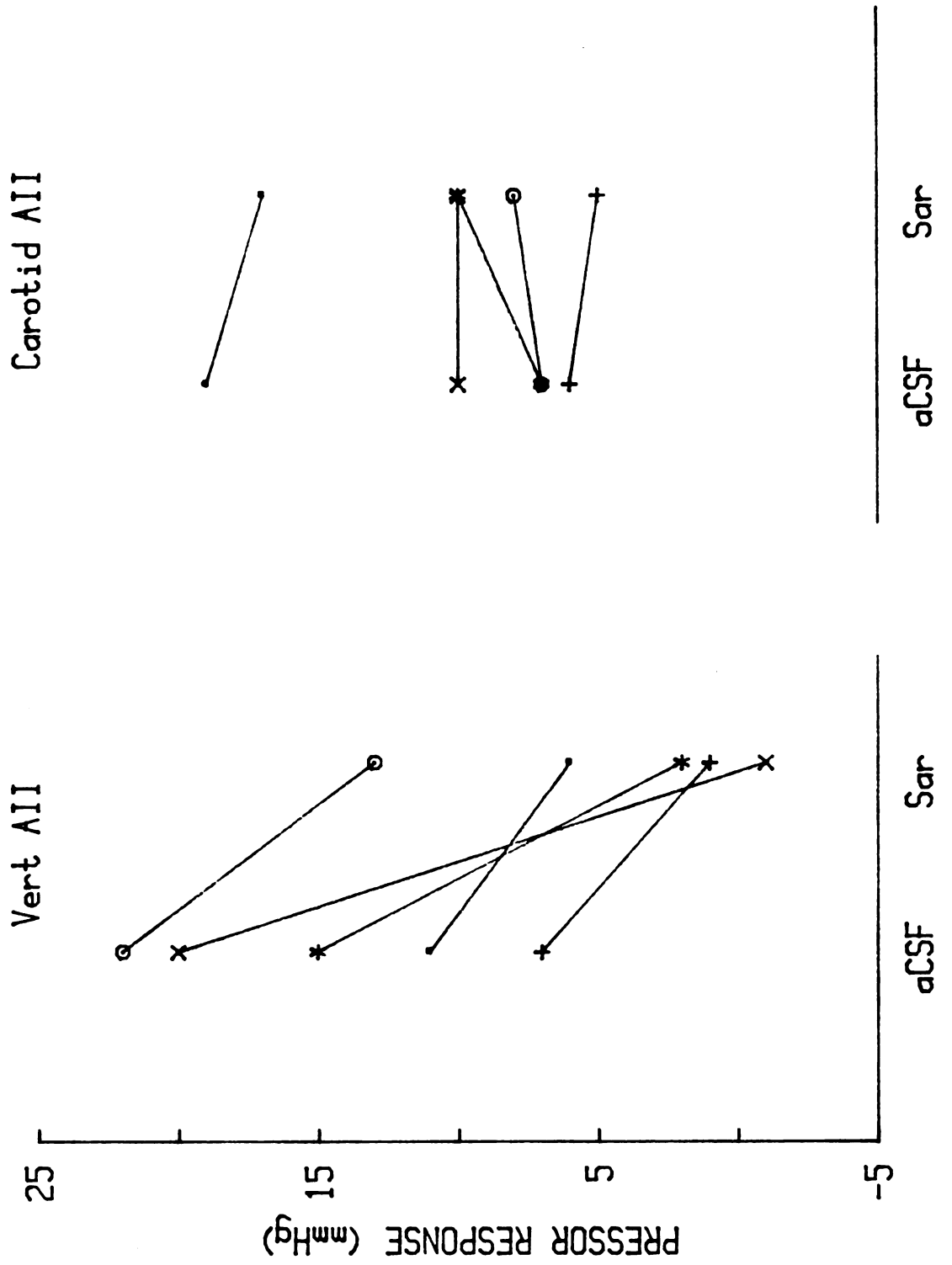


Fig.VI.3 Effects of intraventricular saralasin on the heart rate responses to intravertebral (left) and intracarotid AII in 5 dogs. Saralasin significantly reduced the increase in heart rate following intravertebral AII ($p < 0.05$). The doses of AII were the same as those in the previous figure. The symbols of two dogs are overlapped in the right panel.

Fig. VI.3

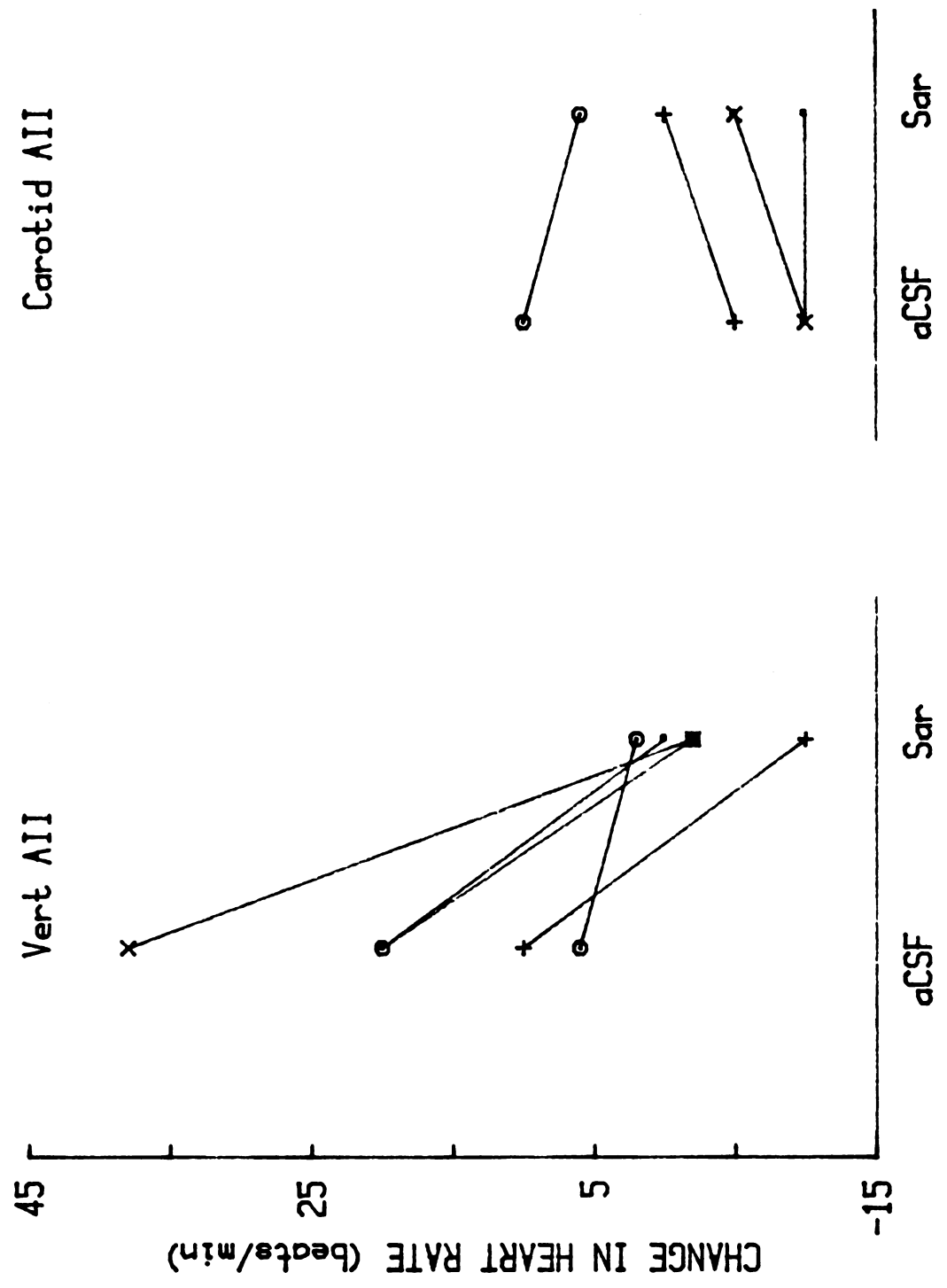
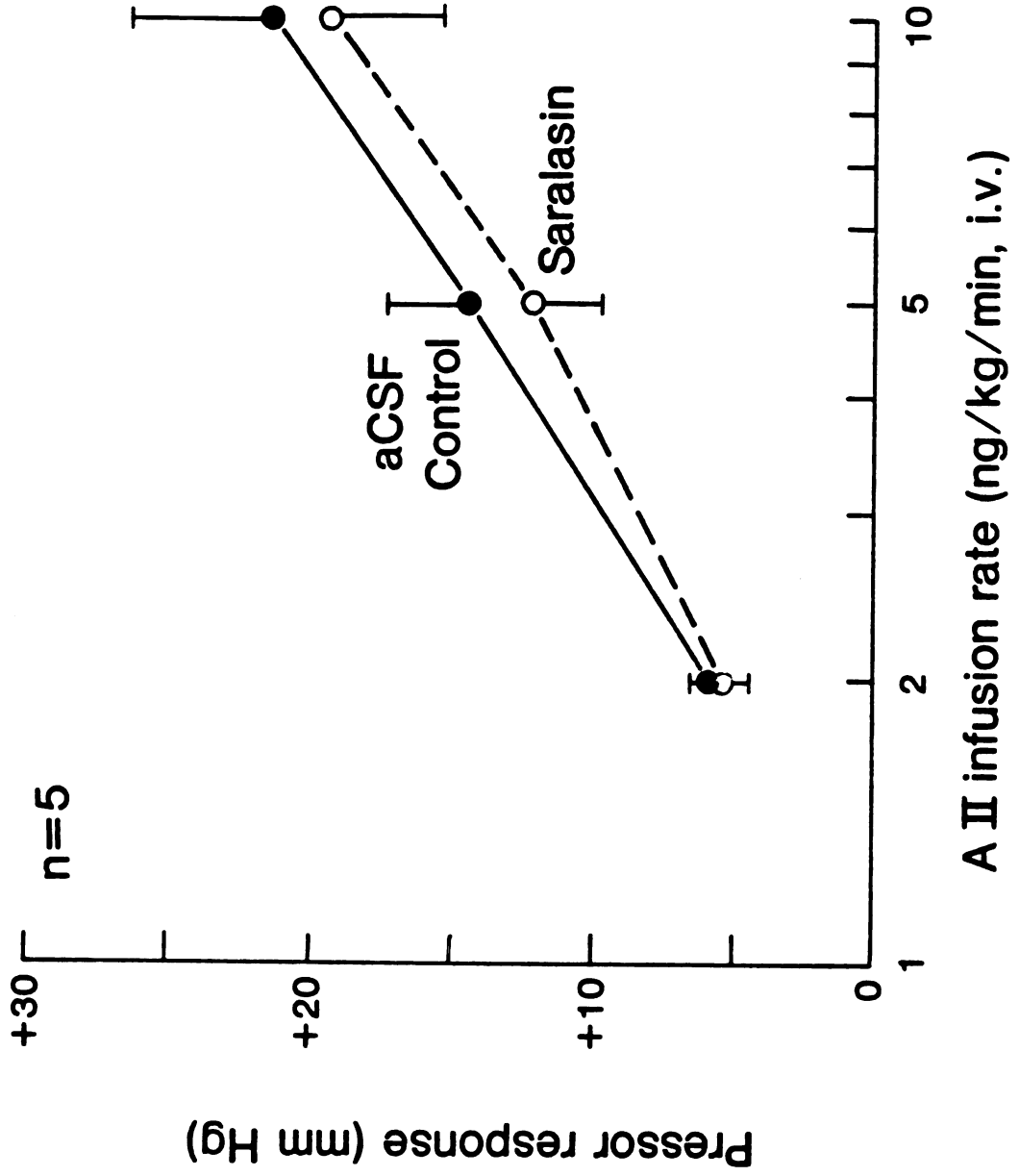


Fig.VI.4 Lack of effect of intraventricular saralasin on the pressor response to i.v. AII. Mean and standard error of mean are shown.

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Fig. VI.4



CHAPTER VII IS ANGIOTENSIN ESSENTIAL IN DRINKING INDUCED
BY WATER DEPRIVATION AND CAVAL LIGATION?*

INTRODUCTION

Angiotensin II has been shown to be a potent dipsogen (Epstein 1978, Fitzsimons 1978), however the role of endogenous angiotensin in the control of drinking is still not established. Ligation of the inferior vena cava was first demonstrated by Fitzsimons (1969) to elicit drinking in the rat. Because this drinking response was inhibited by bilateral nephrectomy (Fitzsimons 1969) and restored by intravenous infusion of AII (Fitzsimons & Simons 1969), he proposed that the renal renin-angiotensin system played a crucial role in the control of drinking following caval ligation. Nevertheless, this hypothesis has been subsequently questioned by several investigators (Lehr et al. 1975, Rolls & Wood 1977, Stricker 1977). Water deprivation is another thirst stimulus that has received much attention. Intracerebroventricular (ICV) administration of saralasin failed to show any inhibition of drinking induced by water deprivation in rats, dogs, sheep, and goats (Abraham et al. 1976, Olsson 1975,

*Published in Am. J. Physiol. 240 (Regulatory Integrative Comp. Physiol. 9): R75-R80, 1981.

1. Introduction

The purpose of this report is to analyze the impact of the COVID-19 pandemic on the global economy and to propose effective strategies for recovery. The report is structured as follows:

- 2. Background
- 3. Methodology
- 4. Results
- 5. Conclusion

2. Background

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, emerged in late 2019 and rapidly spread across the globe. It has led to a significant economic downturn, with many countries experiencing a sharp decline in GDP and high unemployment rates. The World Health Organization (WHO) declared it a global health emergency in January 2020.

3. Methodology

This report uses a combination of secondary data analysis and expert interviews. The data sources include World Bank reports, International Monetary Fund (IMF) publications, and various news articles. The expert interviews were conducted with economists and public health officials to gain insights into the economic and health impacts of the pandemic.

4. Results

The analysis shows that the global economy has experienced a severe contraction. The IMF estimates that the global economy contracted by 3.5% in 2020, with some countries like the United States and China showing a partial recovery in 2021. However, the recovery is uneven, with many developing countries still facing significant challenges. The unemployment rate has risen sharply, and many businesses have closed or reduced operations.

5. Conclusion

The COVID-19 pandemic has had a profound impact on the global economy. To achieve a sustainable recovery, governments and international organizations need to implement coordinated strategies. These include providing financial support to businesses and individuals, investing in infrastructure and healthcare, and promoting digital transformation. Additionally, strengthening global health systems and improving surveillance mechanisms are crucial to prevent future pandemics.

Ramsay & Reid 1975, Severs et al. 1977). However, controversy remains. Malvin, Mouw, and Vander (1977) reported that prolonged infusion of saralasin ICV increased the latency of water-deprived rats to drink, and this has been interpreted as evidence for the participation of the renin-angiotensin system in the control of drinking following water deprivation.

In the present study, we first determined a dose of saralasin ICV that inhibited the drinking responses to AII but not to hypertonic saline. We then used this dose of saralasin to investigate the role of the renin-angiotensin system in the control of drinking following water deprivation and caval ligation.

METHODS

General Methods

Subjects. Male Sprague-Dawley rats (body wt 250-400 g) were used. Except during experiments, animals were housed individually in a room maintained at 20-25°C; lights were on from 5 A.M. to 7 P.M. Water and food (Purina rat chow) were available ad libitum. At 8 A.M. on the day of an experiment, animals were transferred to plastic cages (10 in. x 18 in. x 8 in.) where they had previous experience of all the necessary experimental maneuvers. By 5 P.M. the experiment was

finished, and animals were moved back to their home cages. Consecutive experiments on the same animal were usually separated 2 days apart to minimize any possible carry-over effect.

Chemicals and plasma renin activity assay. Saralasin and angiotensin II were freshly prepared in isotonic saline to the concentration desired, then divided into small volumes and stored at -25°C until needed.

Blood sample were collected by decapitation of the rats. Plasma renin activity (PRA) was measured with a radioimmunoassay for angiotensin I (Reid et al. 1972, Stockigt et al. 1971) and expressed as nanograms of angiotensin I formed per milliliter of plasma during a 3-h incubation.

Intracranial cannulation and infusion. The implantation of lateral ventricular cannula was made according to the procedures of Simpson et al. (1978). At least 4 days were allowed for animals to recover from the surgery. The placement of the cannula was checked by the drinking response to AII ICV (10 ng in 1 μl solution) and rechecked at postmortem by the presence of bromphenol blue inside the ventricle following ICV injection of the dye (1 μl).

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it more precisely. This involves determining the scope of the problem, its causes, and its effects. A clear definition of the problem is essential for developing an effective solution.

2. The second step is to gather information about the problem. This can be done through a variety of methods, including interviews, surveys, and data analysis. The goal is to collect as much relevant information as possible to understand the problem better. This information should be organized and analyzed to identify patterns and trends. For example, a manager might analyze sales data to see if there are any seasonal fluctuations or if certain products are performing better than others.

3. The third step is to generate potential solutions. This is often done through brainstorming or other creative techniques. The goal is to come up with as many possible solutions as possible, without worrying about whether they are realistic or feasible. Once a list of potential solutions is generated, the next step is to evaluate them. This involves comparing the solutions against the problem and the organization's resources and capabilities. The goal is to identify the most promising solution or solutions.

4. The fourth step is to implement the chosen solution. This involves developing a plan of action and putting it into practice. The plan should include a timeline, a budget, and a list of responsibilities. It is important to communicate the plan to all relevant stakeholders and to monitor progress regularly. If the solution is not working, it may be necessary to adjust the plan or try a different solution.

5. The final step is to evaluate the results of the solution. This involves comparing the current performance with the desired state or goal. The goal is to determine whether the solution has been effective and to identify any areas for improvement. If the solution is successful, it should be documented and shared with other managers. If it is not successful, the process should be repeated.

The following regime of administration of saralasin ICV was adopted throughout the study unless otherwise noted. One microliter of saralasin (4 mg/ml) was given ICV as a bolus and followed by infusion at a rate of 1.3 μ l/h for 75 min without any water or food present. The thirst stimulus was then administered with water, but no food, available. Saralasin ICV infusion was continued throughout the rest of the experiment. Animals were unrestrained during the infusion.

Evaluation of Blockade of Angiotensin with Central Saralasin Technique

To verify the capability of saralasin ICV to specifically inhibit drinking to AII, three procedures were carried out. First, drinking was measured for 30 min following an injection of 10 ng AII (10 ng/ μ l) ICV in a group of eight rats treated with saralasin or saline vehicle ICV. Four rats received saralasin first, the other four, saline first. Second, drinking was measured for 30 min following a 20-min i.v. infusion of AII (5 ng/ μ l), via an indwelling jugular catheter (Hsiao et al. 1977), at 100 ng/min (n = 11) in the presence of saralasin or saline ICV. Finally, drinking was measured for 3 h following an intraperitoneal (ip) injection of 1% body weight of 1 M NaCl in

the presence of saralasin (n = 10) or saline vehicle ICV (n = 7). The hypertonic saline was injected under light ether anesthesia.

Water Deprivation

Twenty-four-hour water deprivation. The effect of saralasin ICV on drinking following 24-h water deprivation was studied on 15 rats. They were deprived of water for 24 h with food available. The animals were then randomly divided into two groups, the experimental group (n = 8) received saralasin ICV treatment and the control group (n = 7) received saline vehicle ICV. After the preinfusion of saralasin or saline ICV, animals were allowed free access to water but not food. Water intake was monitored for the following 3 h.

After being deprived of water for 24 h an additional group of nine rats was killed and blood collected for PRA assay. Blood samples were also obtained from a further eight rats for control measurements.

Thirty-hour water deprivation. Fourteen rats were deprived of water for 30 h and were randomly divided into two equal groups. The experimental group received treatment of saralasin ICV and the control group received artificial cerebrospinal fluid (CSF). The composition of the artificial cerebrospinal fluid was

1. Introduction
The purpose of this report is to analyze the impact of the COVID-19 pandemic on the global economy. The report will focus on the economic challenges faced by various countries and the role of international organizations in providing support and guidance.

2. Background
The COVID-19 pandemic began in late 2019 and spread rapidly across the globe. It has caused significant economic disruption, leading to a global recession. Many countries have implemented strict lockdown measures to contain the virus, which has resulted in a sharp decline in economic activity. The World Health Organization (WHO) declared the pandemic a global health emergency in January 2020. The International Monetary Fund (IMF) has estimated that the global economy will contract by 3.5% in 2020, with a projected recovery in 2021.

3. Impact on the Global Economy
The COVID-19 pandemic has had a profound impact on the global economy. It has led to a sharp decline in economic activity, with many countries experiencing a recession. The global economy is expected to contract by 3.5% in 2020, with a projected recovery in 2021. The impact has been particularly severe in emerging and developing economies, which have limited resources to cope with the crisis.

4. Role of International Organizations
International organizations have played a crucial role in providing support and guidance to countries affected by the COVID-19 pandemic. The WHO has coordinated global efforts to contain the virus and has provided technical assistance to countries. The IMF has provided financial support and policy advice to countries facing economic challenges. The World Bank has also provided financial support and policy advice to countries. These organizations have been instrumental in helping countries navigate the crisis and recover from its impact.

5. Conclusion
The COVID-19 pandemic has had a significant impact on the global economy. It has led to a sharp decline in economic activity and a global recession. International organizations have played a crucial role in providing support and guidance to countries affected by the crisis. The global economy is expected to recover in 2021, but the impact of the pandemic will be felt for some time.

described by Malvin et al. (1977). Saralasin was prepared in artificial CSF; the concentration was 20 ng/ μ l. No initial ICV injection was given, and the ICV infusion rate was 200 μ l/h or 4 μ g/h. Seventy-five minutes after the commencement of ICV infusion, water but not food was made available to the animal. Water intake was monitored for the following 90 min.

An additional 16 rats were also deprived of water for 30 h and were randomly divided into two equal groups. The experimental group received treatment with saralasin ICV, and the control group received saline vehicle ICV. The intracranial infusion protocol for 24-h water deprivation was followed, except that the concentration of saralasin was 10 times greater, i.e., 40 μ g/ μ l. Water intake was recorded for 90 min.

Caval Ligation

The abdominal vena cava of a rat was exposed through a small loin incision during ether anesthesia, the cava was then ligated with a silk ligature just above the entry of the right renal vein as described by Fitzsimons (1969). After the incision had been closed, the animal was returned to its cage and the ICV infusion was started immediately. The whole procedure took 10-15 min to complete. Animals were assigned randomly

1. The first part of the text discusses the importance of maintaining accurate records in a laboratory setting. It emphasizes that proper record-keeping is essential for ensuring the reliability and reproducibility of experimental results. The text notes that without accurate records, it would be difficult to identify and troubleshoot any issues that may arise during the course of an experiment.

2. The second part of the text describes the various methods used to collect and analyze data in a laboratory. It highlights the importance of using standardized protocols and procedures to ensure that data is collected consistently and accurately. The text also discusses the use of statistical analysis to interpret the results of experiments and to determine the significance of any observed differences.

3. The third part of the text discusses the importance of safety in a laboratory setting. It emphasizes that safety is a top priority and that all laboratory personnel must be trained in proper safety procedures. The text notes that safety incidents can have serious consequences and that it is essential to take all necessary precautions to prevent such incidents from occurring.

4. The fourth part of the text discusses the importance of communication in a laboratory setting. It emphasizes that clear and concise communication is essential for ensuring that all laboratory personnel are aware of the current status of experiments and any changes to protocols or procedures. The text also discusses the importance of documenting all laboratory activities and results in a clear and concise manner.

Continued on next page

5. The fifth part of the text discusses the importance of quality control in a laboratory setting. It emphasizes that quality control is essential for ensuring that all laboratory results are accurate and reliable. The text notes that quality control procedures should be implemented at all stages of the laboratory process, from the collection of samples to the analysis of results.

6. The sixth part of the text discusses the importance of staying current in a laboratory setting. It emphasizes that laboratory personnel must stay up-to-date on the latest research and techniques in their field. The text notes that staying current is essential for ensuring that laboratory results are accurate and reliable and that it is essential to attend to all necessary training and education.

to receive ICV treatment of either saralasin or saline vehicle. One microliter of the solution was administered as a bolus followed by infusion at a rate of $1.3 \mu\text{l/h}$ for 5.5 or 6 h. In the first series of experiments, 20 animals (body wt 309 ± 7 g) received saralasin ($4 \mu\text{g}/\mu\text{l}$) and the other 14 (320 ± 11 g) received saline. In the second series, 9 rats (311 ± 11 g) received saralasin ($8 \mu\text{g}/\mu\text{l}$) and the other 10 (328 ± 13 g) received saline. Water intake was recorded every 30 min throughout the experiment.

The left carotid arteries of an additional group of rats were cannulated with polyethylene tubing during ether anesthesia. At least 1 day was allowed for animals to recover from the surgery. On the day of the experiment, the arterial catheter was connected to a transducer (Statham, P23Db) and the blood pressure recorded on a polygraph (Grass, model 5). After the blood pressure had remained stable for 10 min, the inferior vena cava of the animal was then ligated and ICV infusion started as described above. Four rats received saralasin ($4 \mu\text{g}/\mu\text{l}$), and the other three received saline vehicle treatment. Blood pressure was monitored continuously for the following 4 h.

The inferior vena cavae of a group of nine rats were ligated, and another group of five were sham

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance against a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it clearly and specifically. This involves determining the scope of the problem, its causes, and its effects. A clear definition of the problem is essential for developing an effective solution.

2. The second step in the process is to analyze the problem. This involves gathering information about the problem and its context. This information can be obtained through various methods, such as interviews, surveys, and data analysis. The goal of this step is to understand the underlying causes of the problem and to identify the factors that are contributing to it. This information is then used to develop a plan of action.

3. The third step in the process is to develop a plan of action. This involves identifying the specific steps that need to be taken to solve the problem. The plan should be realistic and achievable, and it should take into account the resources available and the time constraints. Once a plan has been developed, the next step is to implement it. This involves putting the plan into action and monitoring progress.

4. The fourth step in the process is to implement the plan. This involves putting the plan into action and monitoring progress. It is important to track the results of the plan and to make adjustments as needed. This step is often the most challenging, as it requires the organization to change its behavior and to overcome resistance to change.

5. The fifth and final step in the process is to evaluate the results. This involves comparing the actual results against the desired state or goal. This step is important to determine whether the plan was effective and to identify any areas for improvement. If the plan was not effective, the process may need to be repeated.

operated. The sham-operated animal received the same surgical procedures as the ligated one except that the silk ligature around the vena cava was tightened. Two hours after the surgery, animals were killed by decapitation and blood samples collected for PRA assay.

Data Analysis

Student's t-test was employed to analyze the data of the hypertonic saline injection experiment and of plasma renin activity. Two-factor analysis of variance for repeated measurements (Bostrom 1979, Winer 1971) was performed on all other data. A value of 0.05 was taken as the level of significance. Data are expressed as mean+SE.

RESULTS

Evaluation of Blockade of Angiotensin with Central Saralasin Technique

Injection of 10 ng AII into the lateral ventricle stimulated drinking promptly, and the response lasted a maximum of 15 min. As shown in Table VII.1, saralasin ICV inhibited drinking elicited by AII ICV, whereas the order of presentation of ICV treatments had no significant effect on the drinking response ($P > 0.25$).

Intravenous infusion of AII (100 ng/min) for 20

1. The first part of the text discusses the importance of maintaining accurate records of all transactions and activities. It emphasizes that proper record-keeping is essential for ensuring transparency and accountability in financial reporting. This section also highlights the role of internal controls in preventing errors and fraud.

2. The second part of the text focuses on the importance of regular audits and reviews. It explains that these processes are crucial for identifying potential weaknesses in the system and ensuring that all financial data is accurate and reliable. The text also discusses the benefits of external audits in providing an independent assessment of the organization's financial health.

3. The third part of the text addresses the importance of maintaining up-to-date financial statements. It notes that these statements provide a clear and concise overview of the organization's financial performance and are essential for making informed decisions. The text also discusses the importance of disclosing all relevant information in these statements to ensure transparency.

4. The fourth part of the text discusses the importance of maintaining accurate records of all assets and liabilities. It emphasizes that this information is crucial for determining the organization's net worth and for ensuring that all financial obligations are properly accounted for. The text also discusses the importance of regularly updating these records to reflect any changes in the organization's financial position.

5. The fifth part of the text discusses the importance of maintaining accurate records of all income and expenses. It notes that this information is essential for calculating the organization's profit and loss and for determining its tax liability. The text also discusses the importance of regularly reviewing these records to ensure that all transactions are properly recorded and categorized.

6. The sixth part of the text discusses the importance of maintaining accurate records of all financial transactions. It emphasizes that this information is crucial for ensuring the accuracy and reliability of the organization's financial statements. The text also discusses the importance of regularly reviewing these records to identify any potential errors or discrepancies.

min reliably induced drinking. The latency to drink and amount of water consumed varied from animal to animal, but remained rather constant from experiment to experiment for the same animal. Shortly after the commencement of AII i.v. infusion, animals showed some sign of distress, presumably due to the hypertensive effect of AII. After 5-15 min the animals appeared to be aroused and began to drink. Saralasin ICV abolished the drinking response in all but one animal tested, and the effect was highly significant (Table VII.1). The order of presentation of ICV treatments had no effect on drinking ($P > 0.50$).

Animals recovered very rapidly for the light ether anesthesia, during which 1% body weight of 1 M NaCl was injected ip. They started to drink within 30 min after the injection and in general finished drinking within 90 min. Water intake in the saralasin group was not significantly different from that in the saline controls (Table VII.1).

In summary, saralasin ICV at the dose we used inhibited drinking to AII ICV and i.v., but not to hypertonic saline ip. These results suggest that saralasin ICV specifically antagonizes drinking induced by AII and thus appears to a useful tool to assess the involvement of endogenous AII in drinking following

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it clearly and specifically. This involves determining the scope of the problem, its causes, and its effects. A clear definition of the problem is essential for developing an effective solution.

2. The second step in the process is to analyze the problem. This involves gathering information about the problem and its context. This information can be obtained through observation, interviews, and research. The goal of this step is to understand the underlying causes of the problem and to identify the factors that are contributing to it. This information is then used to develop a hypothesis about the cause of the problem.

3. The third step in the process is to generate potential solutions. This involves brainstorming ideas and evaluating them based on their feasibility and effectiveness. This step is often done in a group setting, where team members can share their ideas and provide feedback. The goal of this step is to identify a solution that is both practical and effective.

4. The fourth step in the process is to implement the solution. This involves putting the solution into action and monitoring its progress. This step is often done in a systematic and organized manner, with clear roles and responsibilities assigned to team members. The goal of this step is to ensure that the solution is implemented correctly and that it leads to the desired outcome.

5. The fifth and final step in the process is to evaluate the results. This involves comparing the actual results with the desired outcome and determining whether the solution was effective. This step is often done through a formal evaluation process, such as a cost-benefit analysis or a customer satisfaction survey. The goal of this step is to determine whether the solution was successful and to identify any areas for improvement.

water deprivation and caval ligation.

Water Deprivation

Twenty-four-hour water deprivation. The initial weight was 325 ± 5 g for the saralasin group and 326 ± 9 g for the controls, and the weight losses due to 24-h water deprivation were 27 ± 1 and 23 ± 1 g, respectively. Immediately after water was made available to the water-deprived animals, they started to drink promptly. Most of the drinking was finished in 30 min. As shown in Fig.VII.1, treatment with saralasin ICV had no significant effect on the drinking response ($P > 0.10$).

Plasma renin activity was 14.0 ± 1.4 ng/ml/3h in rats deprived of water for 24 h ($n = 9$) and 7.1 ± 0.7 in controls ($n = 8$). The difference is significant ($P < 0.001$).

Thirty-hour water deprivation. The initial weight was 308 ± 5 g for the saralasin group and 309 ± 6 g for the control group, and the weight losses due to 30-h deprivation of water were 39 ± 1 and 34 ± 2 g, respectively. Saralasin-treated animals behaved no differently from controls with regard to either the latency to drink or the amount of water consumed (Fig.VII.2, $P > 0.10$).

Even when treated with a much larger dose of saralasin (52 $\mu\text{g}/\text{h}$), another group of animals showed no evidence of suppression of water intake after being deprived of water for 30 h (Fig.VII.3, $P > 0.10$).

Caval Ligation

As shown in Fig.VII.4, for the saline vehicle controls the mean arterial pressure (MAP) dropped to 37 ± 7 mmHg immediately after ligation of the abdominal vena cava. The blood pressure recovered rapidly and reached 90 mmHg in 30 min, by that time animals had already recovered from ether. MAP remained around 100 mmHg for the next 3 h. Treatment with saralasin ICV had no significant effect on the recovery of MAP.

Two hours after caval ligation, plasma renin activity was 34.6 ± 2.9 ng/ml/3h ($n = 9$), whereas it was 14.8 ± 2.1 in the sham-operated group ($n = 5$). The difference is significant ($P < 0.001$).

As shown in Fig.VII.5, most of the drinking occurred in the first 3 h following caval ligation. Treatment with saralasin ICV did not significantly affect water intake in the 5.5-h period following the ligation ($P > 0.10$). When the dose was doubled, saralasin had no significant effect on this drinking response either (Fig.VII.6, $P > 0.25$).

• **Wiederholungsfragen** sind Fragen, die in der Vorlesung oder in den Vorlesungsmaterialien bereits behandelt wurden. Diese Fragen sind oft einfacher zu beantworten, da Sie sich auf das Gelernte beziehen. Sie können jedoch auch dazu beitragen, Ihr Verständnis zu vertiefen und sicherzustellen, dass Sie alle wichtigen Punkte der Vorlesung verstehen.

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• **Wiederholungsfragen** sind Fragen, die in der Vorlesung oder in den Vorlesungsmaterialien bereits behandelt wurden. Diese Fragen sind oft einfacher zu beantworten, da Sie sich auf das Gelernte beziehen. Sie können jedoch auch dazu beitragen, Ihr Verständnis zu vertiefen und sicherzustellen, dass Sie alle wichtigen Punkte der Vorlesung verstehen.

DISCUSSION

As shown in Table VII.1, we have demonstrated that infusion of saralasin inhibited drinking to either AII ICV or i.v., but not to hypertonic saline ip. Because hypertonic saline loading is believed to cause cellular dehydration thirst, which is not mediated by the renin-angiotensin system (Abdelaal et al. 1976, Fitzsimons 1972, Simpson et al. 1978, Summy-Long & Severs 1974), our observations thus suggest that saralasin ICV at the dose we chose (5.2 $\mu\text{g/h}$) is capable of specifically antagonizing drinking induced by AII. As the CSF volume of a rat is no greater than 1 ml, the CSF AII concentration in the ventricle would reach at least 10,000 pg/ml following the injection of 10 ng AII ICV. According to Mann et al. (1980), the plasma AII level would reach 2,000 pg/ml following AII i.v. infusion at a rate of 100 ng/min in a 300-g rat as in this study. This concentration of plasma AII is much higher than those that would have occurred with the other thirst stimuli investigated in the present study, i.e., water deprivation and caval ligation (Abdelaal et al. 1976, Johnson et al. 1981). Thus, if the renin-angiotensin system is involved, treatment with saralasin ICV should show inhibition in water intake following water deprivation and caval ligation.

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it more precisely. This involves determining the scope of the problem, its causes, and its effects. For instance, a manager might define a problem as "a 10% decrease in sales over the last quarter, primarily due to a loss of market share in the competitive market." This definition helps to narrow down the focus of the problem and provides a clear starting point for further investigation.

2. The second step in the process is to gather information about the problem. This involves collecting data and facts that are relevant to the problem. For example, a manager might gather data on sales trends, market conditions, and customer feedback. This information is then analyzed to identify patterns and trends that can help to explain the problem. For instance, a manager might discover that sales are declining because of a new competitor entering the market or because of a change in customer preferences. This information is then used to develop a hypothesis about the cause of the problem.

3. The third step in the process is to develop a hypothesis about the cause of the problem. A hypothesis is a statement that predicts the cause of the problem. For example, a manager might hypothesize that "the decline in sales is due to a loss of market share to a new competitor." This hypothesis is then tested by gathering more information and analyzing it. For instance, a manager might compare sales data for the company and its competitors, or they might conduct a survey of customer preferences. If the hypothesis is supported by the data, then it is likely that the hypothesis is correct. If not, then the manager will need to develop a new hypothesis.

4. The fourth step in the process is to develop a solution to the problem. This involves identifying the actions that need to be taken to address the problem. For example, a manager might develop a solution that involves increasing marketing efforts, improving customer service, or developing new products. The solution is then implemented, and its effectiveness is monitored. For instance, a manager might track sales and customer satisfaction over time to see if the solution is working. If the solution is not working, then the manager will need to develop a new solution.

5. The fifth and final step in the process is to evaluate the solution. This involves determining whether the solution has effectively addressed the problem. For example, a manager might evaluate the solution by comparing sales and customer satisfaction before and after the solution was implemented. If the solution has been effective, then the problem has been solved. If not, then the manager will need to go back to the beginning of the process and start over.

Saralasin ICV however neither increased the latency to drink nor decreased the amount of water consumed in rats deprived of water for 24 h (Fig.VII.1). Malvin et al. (1977), in contrast, reported that infusion of saralasin ICV significantly affected the latency to drink but not the total amount of water drunk in rats deprived of water for 30 h. This has been interpreted as evidence for the participation of the renin-angiotensin system in the control of drinking following water deprivation. As the differences between our results and Malvin et al. were so striking, we tried to repeat their protocol, and the result was again negative (Fig.VII.2), as was the result of another experiment using an even higher dose of saralasin (Fig.VII.3). Whereas the discrepancy between the finding of Malvin et al. and ours is difficult to reconcile, our observations are in line with other reports in rats (Severs et al. 1977), dogs (Ramsay & Reid 1975), goats (Olsson 1975), and sheep (Abraham et al. 1976). The amount of water drunk by the control group in the present study was similar to those reported elsewhere (Hoffman et al. 1978, Ramsay et al. 1977a, Severs et al. 1977) suggesting that the ICV infusion had no deleterious effects on drinking behavior.

As demonstrated in both dogs and rats by Ramsay et al. (1977a & b), approximately two-thirds of water intake following 24-h water deprivation is due to cellular dehydration and the other third due to extracellular dehydration. Because the renin-angiotensin system is not involved in cellular dehydration thirst, it could contribute to, at most, one-third of the drinking induced by water deprivation. Our three sets of consistent observations further indicate that the renin-angiotensin system alone does not play an essential role in the control of drinking following 24- or 30-h water deprivation in rats. However, the possibility that angiotensin combined with another system, e.g., cholinergic system, might subserve this drinking response is not ruled out (Hoffman et al. 1978).

Based primarily on the finding that water intake following ligation of the inferior vena cava was reduced by bilateral nephrectomy (Fitzsimons 1969) and restored by AII i.v. (Fitzsimons & Simons 1969) in the rat, Fitzsimons proposed that the renin-angiotensin system played an important role in the control of drinking following caval ligation. Historically this was the first piece of evidence implicating the renin-angiotensin system in extracellular dehydration thirst (Fitzsimons 1978). However, Stricker (1977, 1978)

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2. The second step in the process is to analyze the problem. This involves gathering data and information about the problem and its causes. This can be done through a variety of methods, including interviews, surveys, and data analysis. For example, a manager might conduct interviews with sales staff to learn more about customer feedback and market trends. Alternatively, the manager might analyze sales data to identify patterns and trends. The goal of this step is to gain a deeper understanding of the problem and its underlying causes. This information is then used to develop a plan of action to address the problem.

reported that nephrectomized rats were in hypotensive shock after caval ligation, presumably due to the absence of the pressor action of AII. He therefore argued that it was the general debility, rather than the removal of the dipsogenic action of AII per se, that rendered the reduction in water intake following caval ligation in nephrectomized rats.

A definitive way to resolve this controversy seems to utilize a treatment that inhibits the dipsogenic action of AII while leaving its pressor effect intact. As shown in Fig.VII.4, saralasin ICV did not affect the recovery of blood pressure following caval ligation. In contrast, as discussed earlier, this dose of saralasin specifically antagonized drinking stimulated by high titers of AII. When applied to caval-ligated rats, however, saralasin ICV did not significantly affect the drinking response (Fig.VII.5). The amount of water drunk by the saline controls is compatible with other studies (Lehr et al. 1975, Stricker 1977), therefore ICV infusion did not affect the drinking response. Even with a larger dose, saralasin still had no significant effect on this drinking (Fig.VII.6). In agreement with Stricker (1977, 1978), Rolls and Wood (1977), and Lehr et al. (1975), these results suggest that the renin-angiotensin system is not essential in

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4. The fourth step in the process is to develop a solution to the problem. This involves identifying the actions that need to be taken to address the problem. For example, a manager might develop a solution that involves increasing marketing efforts, improving customer service, or developing new products. The solution is then implemented and its effectiveness is monitored. For instance, a manager might track sales data and customer feedback to see if the solution is having the desired effect. If the solution is not working, then the manager may need to revise it or develop a new one.

5. The fifth and final step in the process is to evaluate the results of the solution. This involves comparing the current performance with the desired state or goal to see if the problem has been solved. For example, a manager might evaluate the results of a solution by comparing sales data and customer feedback before and after the solution was implemented. If the problem has been solved, then the manager can celebrate the success and move on to other tasks. If the problem has not been solved, then the manager may need to start the process over again.

the control of drinking following caval ligation in rats.

The regulation of blood pressure after ligation of the inferior vena cava poses an intriguing question. The mean arterial pressure recovered from 40 to reach 90 mmHg in 30 min following the ligation (Fig.VII.4). However Stricker (1978) reported that it took 4 h for the blood pressure of caval-ligated rats to get to the same level. The difference is probably due to anesthesia; our observations were made on animals which had recovered from ether by 30 min after the ligation, whereas Stricker's were made on rats during pentobarbital anesthesia throughout the entire experimental period.

In summary, infusion of saralasin ICV at a proper dose specifically antagonized drinking induced by AII, however it did not inhibit water intake following water deprivation or caval ligation. These findings indicate that the renin-angiotensin system alone does not play an essential role in the control of drinking following water deprivation or caval ligation in rats.

Table VII.1 Evaluation of blockade of angiotensin with central saralasin technique

stimulus	n	Saralasin ICV	Saline ICV	P
AII ICV	8	0.5 \pm 0.3*	6.9 \pm 1.3*	<0.005
AII i.v.	11	0.09 \pm 0.09*	3.3 \pm 0.5*	<0.001
1 M NaCl ip	17	2.7 \pm 0.3# (n = 10)	2.2 \pm 0.5# (n = 7)	>0.40

*Water intake (ml) in 30 min following the challenge of the thirst stimulus. #Water intake (ml/100 g body wt) in 90 min following the challenge of the thirst stimulus.



Fig.VII.1 Effect of administration of saralasin ICV (5.2 $\mu\text{g}/\text{h}$) on water intake following 24-h water deprivation. There was no significant difference between saralasin group (n = 8) and saline vehicle controls (n = 7) ($P > 0.10$). Water was made available at t = 0. Mean and SE are shown.

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Fig. VII.1

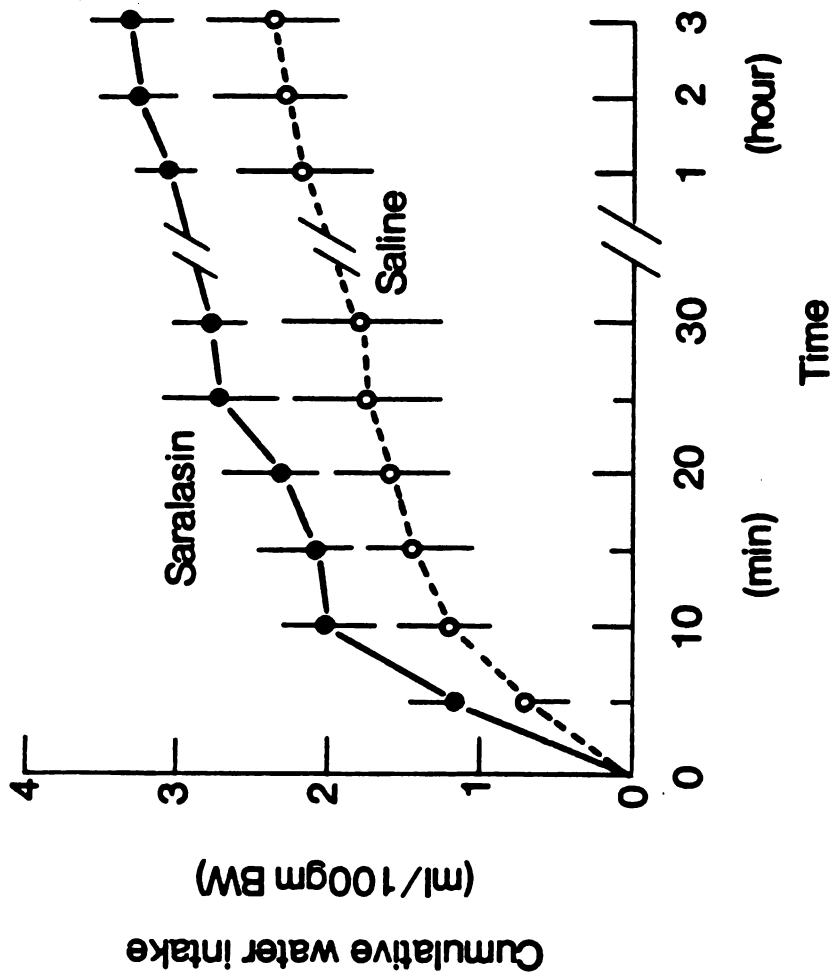


Fig.VII.2 Effect of administration of
saralasin ICV (4.0 $\mu\text{g}/\text{h}$) on water intake following 30-h
water deprivation. Protocol of Malvin et al. (1977)
for saralasin treatment was followed. There was no
significant difference between saralasin group and CSF
controls ($P > 0.10$). Water was made available at $t =$
0. Mean and SE are shown. $n = 7$ in both groups.

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Fig. VII.2

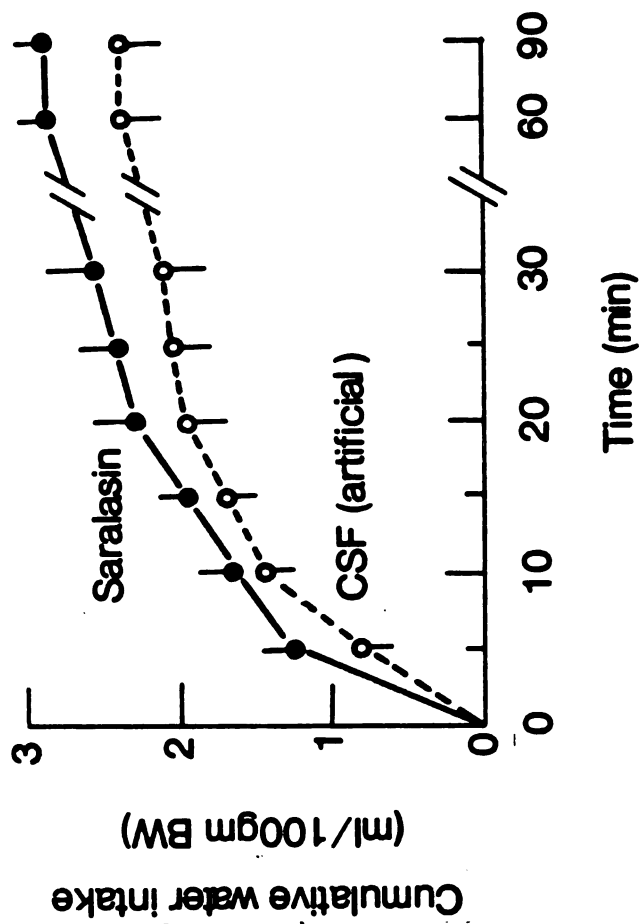


Fig.VII.3 Effect of administration of saralasin ICV (52 $\mu\text{g}/\text{h}$) on water intake following 30-h water deprivation. Difference between saralasin group and saline vehicle controls was not significant ($P > 0.10$). Water was made available at $t = 0$. Mean and SE are shown. $n = 8$ in both groups.

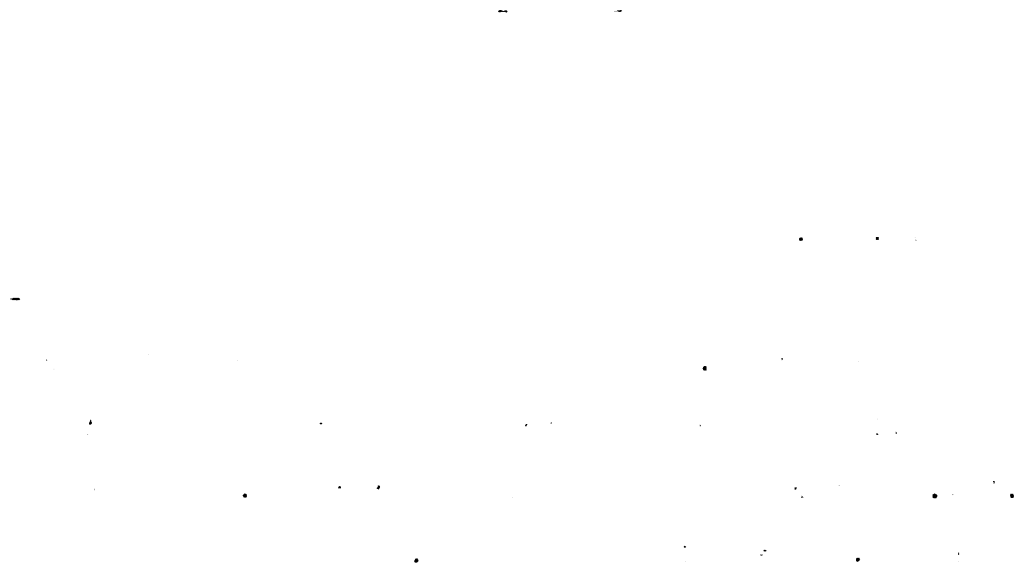


Fig. VII.3

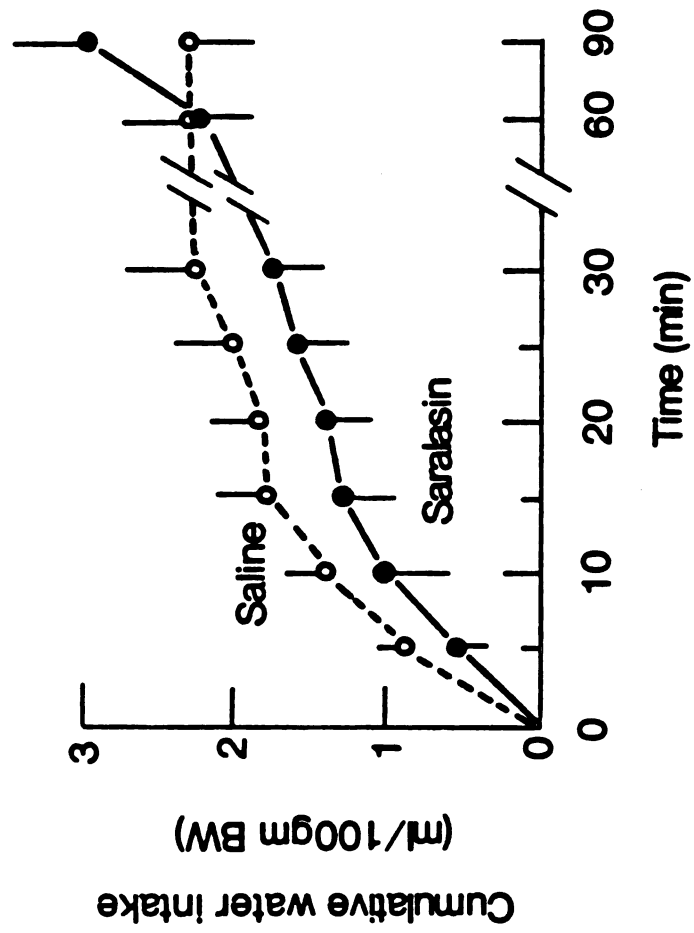


Fig.VII.4 Effect of saralasin ICV (5.2 $\mu\text{g/h}$) on recovery of blood pressure following caval ligation (t = 0). No significant difference existed between saralasin group (n = 4) and saline vehicle controls (n = 3). Mean and SE are shown.

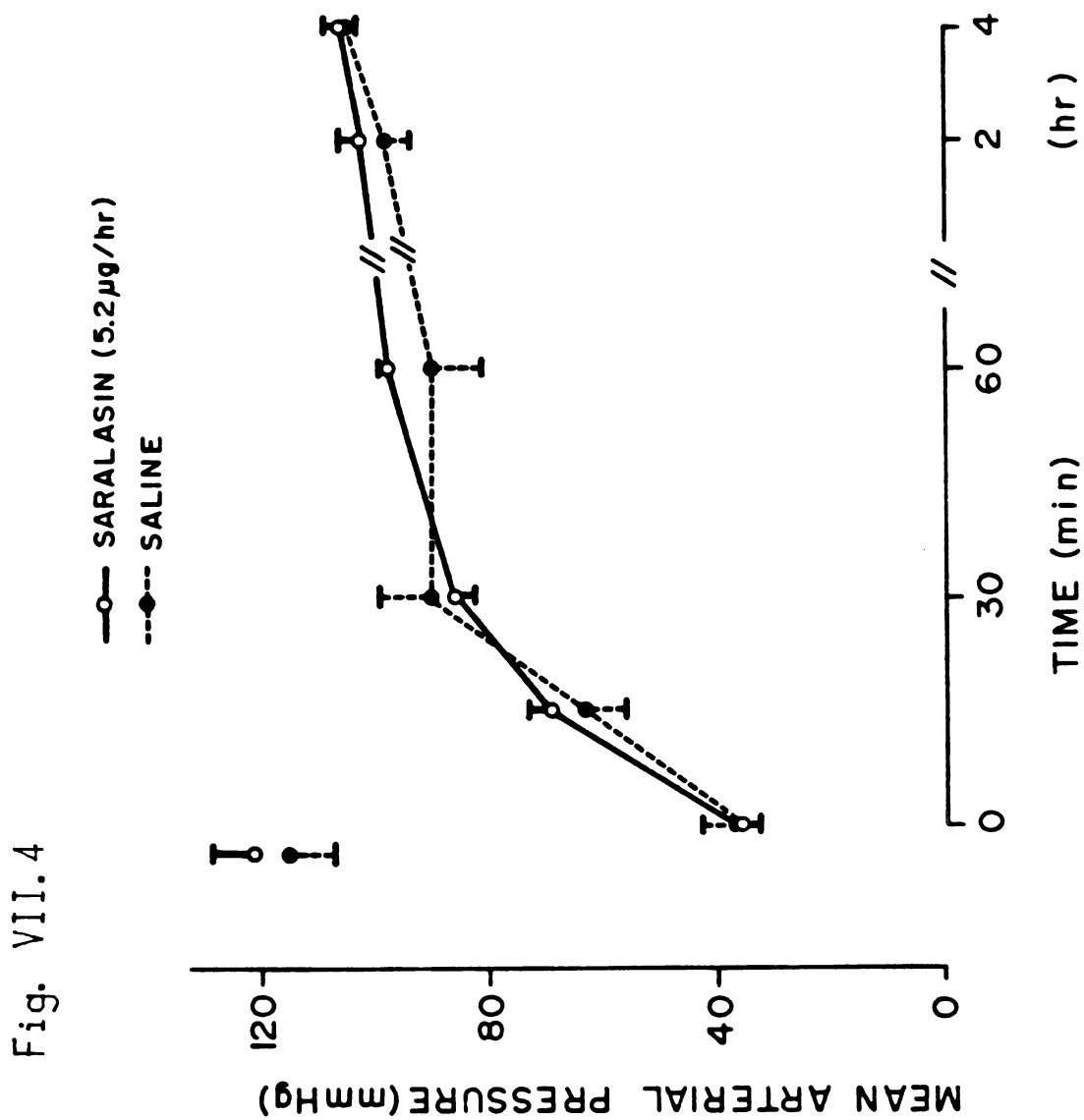


Fig. VII.4

Fig.VII.5 Effect of administration of saralasin ICV (5.2 $\mu\text{g}/\text{h}$) on water intake following caval ligation (t = 0). Difference between saralasin group (n = 20) and saline vehicle controls (n = 14) was not significant (P > 0.10). Mean and SE are shown.

1. The first part of the document discusses the importance of maintaining accurate records of all transactions and activities. It emphasizes that proper record-keeping is essential for ensuring transparency and accountability in financial reporting.

2. The second part of the document outlines the various methods and techniques used to collect and analyze data. It highlights the need for consistent and reliable data sources to support the findings of the study.

3. The third part of the document presents the results of the analysis, showing that there is a significant correlation between the variables studied. This finding suggests that the factors being investigated have a strong influence on the outcomes.

4. The fourth part of the document discusses the implications of the findings and provides recommendations for future research. It suggests that further studies should be conducted to explore the underlying mechanisms and to test the findings in different contexts.

5. The fifth part of the document concludes the study and summarizes the key findings. It reiterates the importance of accurate record-keeping and the need for rigorous data analysis in financial reporting.

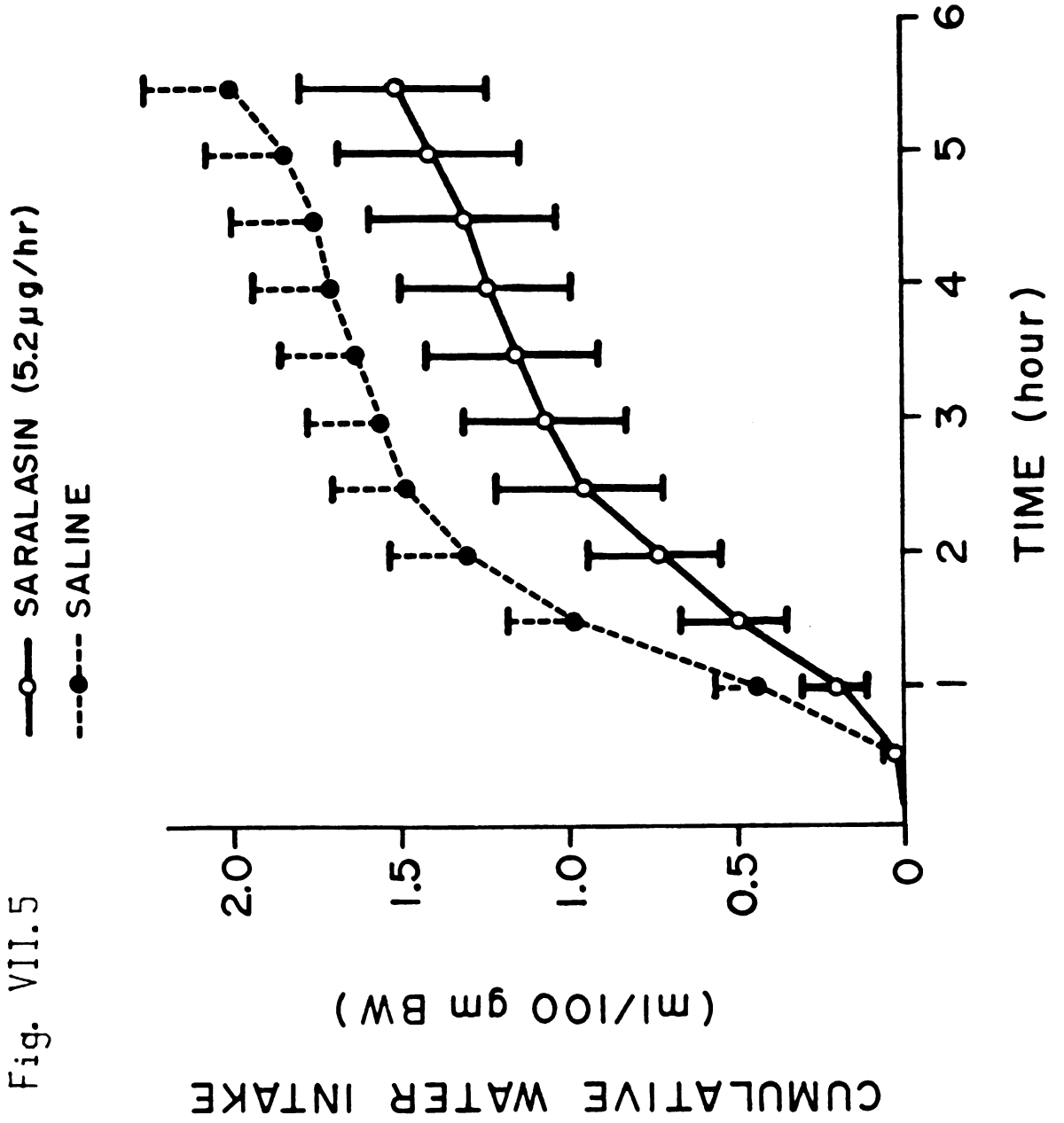


Fig.VII.6 Effect of administration of saralasin ICV (10.4 $\mu\text{g/h}$) on water intake following caval ligation ($t = 0$). Difference between saralasin group ($n = 9$) and saline vehicle controls ($n = 10$) was not significant ($P > 0.25$). Mean and SE are shown.

—○— SARALASIN (10.4 $\mu\text{g/hr}$)
- -●- - SALINE

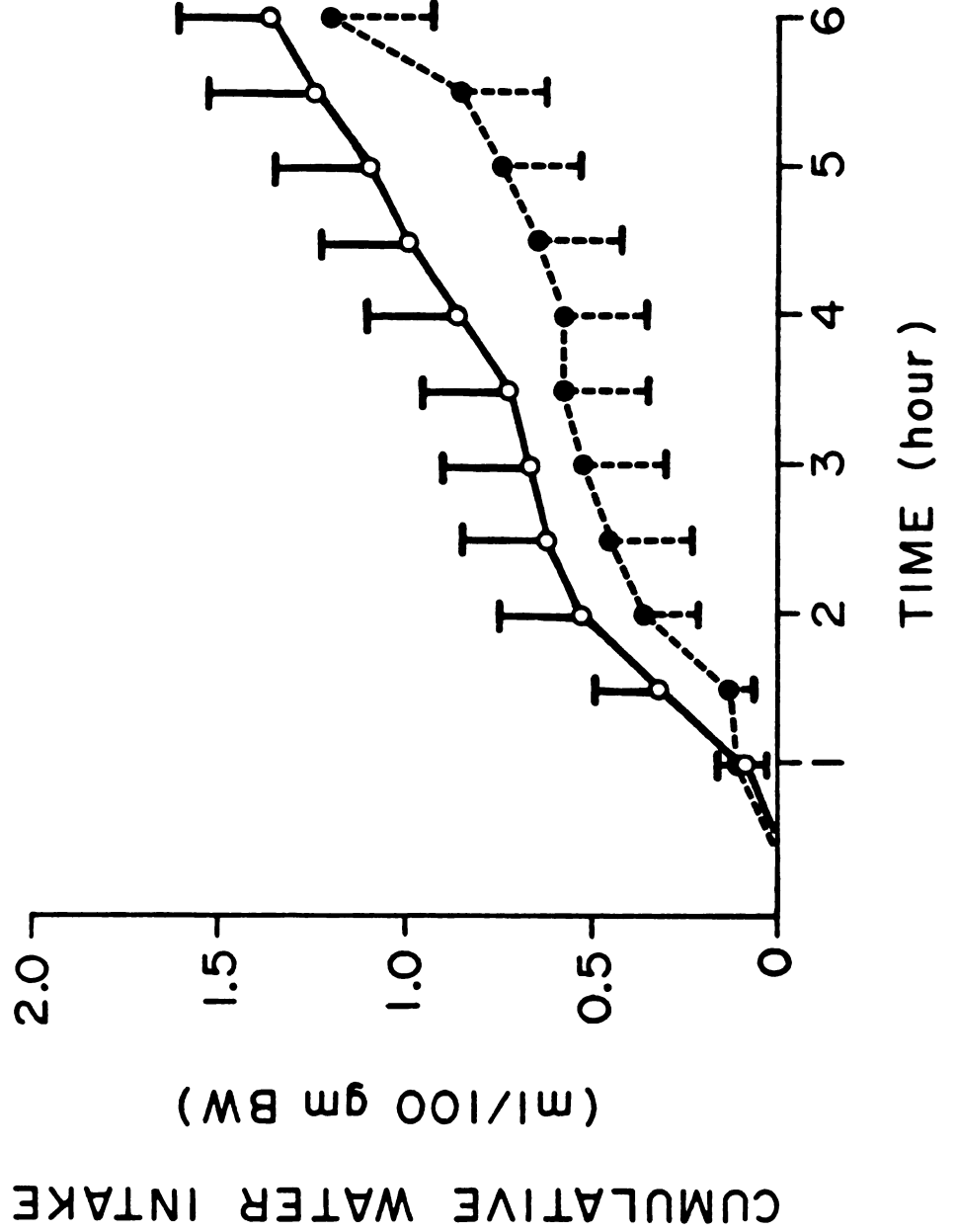


Fig. VII.6

CHAPTER VIII GENERAL DISCUSSION AND CONCLUSIONS

The pressor response to infusion of AII into the common carotid arteries in conscious mongrel dogs is clearly different from that to intravertebral AII. Firstly, the pressor response to intracarotid AII, unlike that to intravertebral AII, is slow in onset and not accompanied by an increase in heart rate. Secondly, the area postrema, located in the caudal medulla, is generally considered to be the site of pressor action of intravertebral AII. Since the carotid blood does not perfuse the medulla (Table IV.1), the site of action of intracarotid AII cannot be the same as that of intravertebral AII. Finally, intraventricular saralasin, a competitive antagonist of AII, inhibited the pressor response to intravertebral, but not to intracarotid, AII (Fig.VI.2). This provides direct evidence that the site of action of intracarotid AII is different from that of intravertebral AII.

However, the mechanism responsible for the pressor effect of intracarotid AII remains unclear. Extracranial vasoconstriction could possibly account for this pressor effect. Intracarotid AII at 1.0 ng/kg/min reduced the carotid blood flow to half (Fig.III.1), which could result in a pressor response of 5 mmHg

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4. The fourth step in the process is to develop a solution to the problem. This involves identifying the actions that need to be taken to address the problem. For example, a manager might develop a solution that involves increasing marketing efforts, improving customer service, or developing new products. The solution is then implemented, and its effectiveness is monitored. For instance, a manager might track sales and customer satisfaction over time to see if the solution is working. If the solution is not working, then the manager will need to re-evaluate the problem and develop a new solution.

5. The fifth and final step in the process is to evaluate the results of the solution. This involves comparing the current performance with the desired state or goal. For example, a manager might evaluate the results of a solution by comparing sales and customer satisfaction to the initial state of the problem. If the results are positive, then the solution is effective. If not, then the manager will need to re-evaluate the problem and develop a new solution. This step is important because it allows the manager to see if the solution is working and to make adjustments if necessary.

according to the theoretical analysis (Fig.III.4). The difference between the pressor effect of intracarotid AII and that of AII i.v. in these experiments happened to be 5 mmHg (Fig.III.3).

The mean blood flow in each internal carotid artery in conscious dogs determined by the radioactive microsphere method was 7.7 ml/min, and that in the anastomotic artery between the external and internal carotid arteries was 3.3 ml/min. Additional evidence exists that anastomotic blood mixed well with internal carotid blood before reaching the cerebral microcirculation (Fig.IV.2). Therefore, although the external carotid artery contributes significantly to the perfusion of the brain, the brain would receive significantly higher concentration of AII when AII was infused into the common carotid artery than when infused into the external carotid artery (Tables IV.1 & 2). This argument is supported by the observation that plasma vasopressin concentration increased more when AII was infused into the common carotid artery than when infused into the external carotid artery (Table IV.4). However, the magnitude of the pressor effect of AII infused into the common carotid artery was the same as that infused into the external carotid artery. These findings therefore argue against the hypothesis that

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the pressor response to intracarotid AII is centrally mediated.

However, it is difficult to explain the finding that plasma corticosteroid concentration increased to the same degree when AII was infused into either common or external carotid artery (Table IV.5). Plasma corticosteroid concentration was used as an index of ACTH secretion. Although it is not very likely, the possibility exists that plasma corticosteroid concentration might not accurately reflect ACTH secretion.

The finding that intracarotid infusions of phenylephrine and vasopressin caused smaller increase in blood pressure than intravenous infusions (Fig.V.1 & 3) seem to be at odds with the extracranial vasoconstriction hypothesis. However, evidence exists that intracarotid vasopressin and phenylephrine could act directly on the carotid sinus to increase baroreceptor activity and therefore result in a smaller pressor response. In contrast, AII appears not to have such action on the carotid sinus since both common and external carotid infusions of AII induced same degree of pressor response (Fig.III.3). Therefore, these findings do not rule out the extracranial vasoconstriction hypothesis.

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal.

2. Once a problem is identified, the next step is to define the problem more precisely. This involves identifying the causes of the problem and the scope of the problem.

3. The third step is to generate potential solutions. This is often done by brainstorming or using a structured problem-solving process.

4. The fourth step is to evaluate the potential solutions. This involves comparing the solutions against the criteria that were used to define the problem.

5. The fifth step is to select a solution. This is often done by choosing the solution that is most likely to be successful and that is most consistent with the organization's values and goals.

6. The sixth step is to implement the solution. This involves putting the solution into action and monitoring its progress.

7. The seventh step is to evaluate the results of the solution. This involves comparing the actual results with the expected results.

8. The eighth step is to adjust the solution if necessary. This involves making changes to the solution if it is not working as well as expected.

9. The ninth step is to document the solution. This involves recording the steps that were taken to solve the problem and the results that were achieved.

10. The tenth step is to share the solution. This involves communicating the solution to others who may be facing a similar problem.

11. The eleventh step is to review the process. This involves reflecting on the steps that were taken and identifying areas for improvement.

12. The twelfth step is to prevent the problem from recurring. This involves identifying the underlying causes of the problem and taking steps to address them.

In conclusion, although some of the evidence suggests that the pressor action of intracarotid AII is due to extracranial vasoconstriction, definite conclusions are not possible.

The central pressor action of intravertebral AII appears not to be essential for the pressor response to blood-borne AII. This is because that intraventricular saralasin inhibited the pressor effect of intravertebral, but not of intravenous, AII (Figs.VI.2 & 4). If the pressor action of intravertebral AII is essential, saralasin should reduce the pressor effect of AII i.v. as well.

Blood-borne AII appears not to play an essential role in drinking following water deprivation and caval ligation in rats. This is because that intraventricular saralasin specifically inhibited drinking to AII i.v.; however, it had no significant effect on drinking following the above two stimuli.

the first part of the book. In the second part, the author discusses the impact of the COVID-19 pandemic on the global economy and the role of the World Bank in providing financial assistance to affected countries. The author also discusses the impact of the pandemic on the global environment and the role of the World Bank in promoting sustainable development. The book is a valuable resource for anyone interested in the role of the World Bank in the global economy.

The book is divided into two main parts. The first part, titled "The World Bank and the Global Economy," discusses the role of the World Bank in the global economy and the impact of the COVID-19 pandemic. The second part, titled "The World Bank and the Global Environment," discusses the impact of the COVID-19 pandemic on the global environment and the role of the World Bank in promoting sustainable development.

The author, [Name], is a senior advisor at the World Bank and has written extensively on the global economy and the environment. The book is a valuable resource for anyone interested in the role of the World Bank in the global economy and the environment.

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REFERENCES

Abdelaal AE, Mercer PF and Mogenson GJ (1976). Plasma angiotensin II levels and water intake following beta-adrenergic stimulation, hypovolemia, cellular dehydration and water deprivation. *Pharmacol. Biochem. Behav.* 4:317.

Abraham SF, Denton DA, McKinley MJ and Weisinger RS (1976). Effect of an angiotensin antagonist, [1-Sar, 8-Ala]-angiotensin II on physiological thirst. *Pharmacol. Biochem. Behav.* 4:243.

Akinkugbe OO, Brown WCB, and Cranston WI (1966). Pressor effects of angiotensin infusions into different vascular beds in the rabbit. *Clin. Sci.* 30:409.

Al-Merani SAMA, Brooks DP, Chapman BJ, and Munday KA (1978). The half-lives of angiotensin II, AII-amide, AIII, saralasin and renin in the circulatory system of the rat. *J. Physiol.* 278:471.

Barnes KL, Ferrario CM, and Conomy JP (1979). Comparison of the hemodynamic changes produced by electrical stimulation of the area postrema and nucleus tractus solitarius in the dog. *Circ. Res.* 45:136.

Bickerton PK and Buckley JP (1961). Evidence for a central mechanism in angiotensin induced hypertension.

1. 1920年11月，在蘇聯政府的支持下，在莫斯科舉行了第一次全蘇蘇維埃代表大會。大會通過了蘇聯憲法，確立了蘇聯的國家體制。

2. 1922年3月，蘇聯政府宣佈成立，標誌著蘇聯的正式成立。

3. 1924年12月，列寧逝世，斯大林繼任蘇聯領導人。

4. 1928年，蘇聯開始實施第一個五年計劃，旨在實現工業化。

5. 1936年，蘇聯通過了新的憲法，確立了社會主義制度。

6. 1939年，蘇聯與德國簽訂了互不侵犯條約，標誌著蘇聯正式加入第二次世界大戰。

7. 1941年6月，德國發動巴巴羅薩行動，蘇聯正式參戰。

8. 1945年，蘇聯在二戰中取得勝利，成為世界超級大國之一。

9. 1946年，蘇聯開始實施第二個五年計劃，進一步加強工業化。

10. 1953年，斯大林逝世，赫魯曉夫繼任蘇聯領導人。

11. 1956年，蘇聯開始實施改革開放政策，旨在提高經濟效率。

12. 1961年，蘇聯成功發射了第一顆人造衛星，標誌著太空時代的開始。

13. 1968年，蘇聯出兵捷克斯拉夫，引發了冷戰期間的蘇聯入侵。

14. 1979年，蘇聯出兵阿富汗，引發了蘇聯入侵阿富汗。

15. 1985年，戈爾巴喬夫繼任蘇聯領導人，開始實施改革開放政策。

16. 1989年，蘇聯開始實施民主化改革，旨在提高政治透明度。

17. 1991年，蘇聯解體，俄羅斯聯邦成立。

18. 1993年，俄羅斯聯邦通過了新的憲法，確立了民主制度。

19. 1995年，俄羅斯聯邦開始實施第三個五年計劃，旨在實現經濟復甦。

20. 1999年，俄羅斯聯邦開始實施第四個五年計劃，進一步加強經濟建設。

21. 2000年，普京繼任俄羅斯領導人，開始實施改革開放政策。

22. 2004年，俄羅斯聯邦開始實施第五個五年計劃，進一步加強經濟建設。

23. 2008年，俄羅斯聯邦開始實施第六個五年計劃，進一步加強經濟建設。

24. 2012年，普京繼任俄羅斯領導人，開始實施改革開放政策。

25. 2014年，俄羅斯聯邦開始實施第七個五年計劃，進一步加強經濟建設。

26. 2018年，俄羅斯聯邦開始實施第八個五年計劃，進一步加強經濟建設。

27. 2020年，俄羅斯聯邦開始實施第九個五年計劃，進一步加強經濟建設。

28. 2022年，俄羅斯聯邦開始實施第十個五年計劃，進一步加強經濟建設。

Proc. Soc. Exp. Bio. Med. 106:834.

Biron P, Koiv E, Nowaczynski W, Brouillet J, and Genest J (1961). The effects of intravenous infusions of valine-5 angiotensin II and other pressor agents on urinary electrolytes and corticosteroids, including aldosterone. J. Clin. Invest. 40:338.

Bostrom A (1979). RMEAS - A program for repeated measures analysis of variance, version 5.0. San Francisco, CA: University of California.

Bouckaert JJ and Heymans C (1935). On the reflex regulation of the cerebral blood flow and the cerebral vaso-motor tone. J. Physiol. 84:367.

Bohr DF (1974). Angiotensin on vascular smooth muscle. In: Angiotensin , Page IH and Bumpus FM (eds.), New York: Springer-Verlag, pp.424-440.

Bonjour JP & Malvin RL (1970). Stimulation of ADH release by the renin-angiotensin system. Am. J. Physiol. 218:1555.

Booth DA (1968). Mechanism of action of norepinephrine in eliciting an eating response on injection into the rat hypothalamus. J. Pharmacol. Exp. Ther. 160:336.

Braun-Menendez E, Fasciolo TC, Leloir LF, and Munoz JM

(1940a). The substance causing renal hypertension. *J. Physiol.* 98:283.

Braun-Menendez E, Fasciolo TC, Leloir LF, and Munoz JM (1940b). *Farmacologia de la hipertensina.* *Rev. Soc. Argent. Biol.* 16:398.

Brightman MW, Klatzo I, Olsson Y, and Reese TS (1970). The blood-brain barrier to proteins under normal and pathological conditions. *J. Neurol. Sci.* 10:215.

Brody MJ, Fink GD, Buggy J, Haywood JR, Gordon FJ, and Johnson AK (1978). The role of the anteroventral third ventricle (AV3V) region in experimental hypertension. *Circ. Res.* 43 (Suppl. I):I2.

Brooks VL, Reid IA, and Keil LC (1980). Effects of angiotensin II on ACTH and vasopressin secretion (Abstract). *Fed. Proc.* 39:946.

Brooks VL and Reid IA (1981). Effects of intravertebral and intracarotid infusion of the angiotensin II antagonist saralasin in conscious, sodium depleted dogs (Abstract). *Fed. Proc.* 40:488.

Buckberg GD, Luck JC, Payne BD, Hoffman JIE, Archie JP and Fixler (1971). Some sources of error in measuring regional blood flow with radioactive microspheres. *J. Appl. Physiol.* 31:598.

Buggy J, Fink GD, Johnson AK, and Brody MJ (1977). Prevention of the development of renal hypertension by anteroventral third ventricular tissue lesions. *Circ. Res.* 40 (Suppl.I):I110.

Bumpus FM and Page IH (1954). Preliminary studies on the structure of angiotonin. *Science* 119:849.

Cash JR (1926). Further studies of arterial hypertension. *Proc. Soc. Exp. Biol. Med.* 23:609.

Cook WF (1971). Cellular localisation of renin. In: *Kidney Hormones*, Fisher JW (ed.), New York: Academic Press, pp.117-128.

Crill WE and Reis DJ (1968). Distribution of carotid sinus and depressor nerves in the cat brainstem. *Am. J. Physiol.* 214:269.

Davis JO (1963). Importance of the renin-angiotensin system in the control of aldosterone secretion. In: *Hormones and the Kidney*, Williams PC (ed.), New York: Academic Press, pp.325-329.

Davis JO (1974). The renin-angiotensin system in the control of aldosterone secretion. In: *Angiotensin*, Page IH and Bumpus FM (eds.), New York: Springer-Verlag, pp.322-336.

的。在图 10-1 中， \mathcal{A} 和 \mathcal{B} 是 \mathcal{A} 和 \mathcal{B} 的任意子集。由图 10-1 可知， $\mathcal{A} \cap \mathcal{B}$ 是 \mathcal{A} 和 \mathcal{B} 的交集， $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{A} 和 \mathcal{B} 的并集。由图 10-1 可知， $\mathcal{A} \cap \mathcal{B}$ 是 \mathcal{A} 和 \mathcal{B} 的交集， $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{A} 和 \mathcal{B} 的并集。

图 10-1 展示了两个集合 \mathcal{A} 和 \mathcal{B} 的交集和并集。图中， \mathcal{A} 和 \mathcal{B} 是两个不相交的集合，它们的交集 $\mathcal{A} \cap \mathcal{B}$ 是空集，而它们的并集 $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{A} 和 \mathcal{B} 的并集。

图 10-2 展示了两个集合 \mathcal{A} 和 \mathcal{B} 的交集和并集。图中， \mathcal{A} 和 \mathcal{B} 是两个相交的集合，它们的交集 $\mathcal{A} \cap \mathcal{B}$ 是非空集，而它们的并集 $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{A} 和 \mathcal{B} 的并集。

图 10-3 展示了两个集合 \mathcal{A} 和 \mathcal{B} 的交集和并集。图中， \mathcal{A} 和 \mathcal{B} 是两个包含关系的集合， \mathcal{A} 包含 \mathcal{B} ，它们的交集 $\mathcal{A} \cap \mathcal{B}$ 是 \mathcal{B} ，而它们的并集 $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{A} 。

图 10-4 展示了两个集合 \mathcal{A} 和 \mathcal{B} 的交集和并集。图中， \mathcal{A} 和 \mathcal{B} 是两个包含关系的集合， \mathcal{B} 包含 \mathcal{A} ，它们的交集 $\mathcal{A} \cap \mathcal{B}$ 是 \mathcal{A} ，而它们的并集 $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{B} 。

图 10-5 展示了两个集合 \mathcal{A} 和 \mathcal{B} 的交集和并集。图中， \mathcal{A} 和 \mathcal{B} 是两个包含关系的集合， \mathcal{A} 包含 \mathcal{B} ，它们的交集 $\mathcal{A} \cap \mathcal{B}$ 是 \mathcal{B} ，而它们的并集 $\mathcal{A} \cup \mathcal{B}$ 是 \mathcal{A} 。

Davis JO and Freeman RH (1976). Mechanisms regulating renin release. *Physiol. Rev.* 56:1.

Deuben RR and Buckley JP (1970). Identification of a central site of action of angiotensin II. *J. Pharmacol. Exp. Ther.* 175:139.

Dutta SN, Davis DA, Booker WM, and Pradhan SN (1971). Responses to microinjections of angiotensin into the hypothalamus of cats. *Neuropharmacology* 10:231.

Ellenberger W and Baum H (1891). *Anatomie des Hundes*. Berlin: Paul Parey.

Elliott DF and Peart WS (1957). The amino acid sequence in a hypertensin. *Biochem. J.* 65:246.

Epstein AN (1978). The neuroendocrinology of thirst and salt appetite. In: *Frontiers in Neuroendocrinology*, Ganong WF and Martini L (eds.), New York: Raven, vol. 5, pp.101-134.

Epstein AN, Fitzsimons JT, and Rolls BJ (1970). Drinking induced by injection of angiotensin into the brain of the rat. *J. Physiol.* 210:457.

Erdo EG (1977). The angiotensin I converting enzyme. *Fed. Proc.* 36:1760.

The first part of the proof shows that \mathbb{Z} is a subring of \mathbb{R} . To do this, we need to verify that \mathbb{Z} is closed under addition, subtraction, and multiplication, and that it contains the additive identity (0) and the multiplicative identity (1).

Let $a, b \in \mathbb{Z}$. Then $a + b \in \mathbb{Z}$, $a - b \in \mathbb{Z}$, and $ab \in \mathbb{Z}$. Also, $0 \in \mathbb{Z}$ and $1 \in \mathbb{Z}$. Therefore, \mathbb{Z} is a subring of \mathbb{R} .

Next, we show that \mathbb{Z} is not a field. For this, we need to find an element in \mathbb{Z} that does not have a multiplicative inverse in \mathbb{Z} . Consider the element 2. Its multiplicative inverse is $\frac{1}{2}$, which is not an integer. Therefore, \mathbb{Z} is not a field.

Finally, we show that \mathbb{Z} is a local ring. A local ring is a ring with a unique maximal ideal. In \mathbb{Z} , the maximal ideals are the prime ideals, which are of the form (p) where p is a prime number. There are infinitely many prime numbers, so \mathbb{Z} does not have a unique maximal ideal. Therefore, \mathbb{Z} is not a local ring.

In conclusion, \mathbb{Z} is a subring of \mathbb{R} , but it is not a field and not a local ring.

Evans HE and Christensen GC (1979). Miller's Anatomy of the Dog. 2nd ed. Philadelphia: Saunders.

Fan F-C, Schuessler GB, Chen RYZ, and Chien S (1979). Determinations of blood flow and shunting of 9- and 15- μ m spheres in regional beds. Am. J. Physiol. 237 (Heart Circ. Physiol. 6): H25.

Feldberg W and Lewis GP (1964). The action of peptides on the adrenal medulla. Release of adrenaline by bradykinin and angiotensin. J. Physiol. 171:98.

Felix D and Akert K (1974). The effect of angiotensin II on neurons of the cat subfornical organ. Brain Res. 76:350.

Ferrario CM, Dickinson CJ, and McCubbin JW (1970). Central vasomotor stimulation by angiotensin. Clin. Sci. 39:239.

Ferrario CM, Gildenberg PL, and McCubbin JW (1972). Cardiovascular effects of angiotensin mediated by the central nervous system. Circ. Res. 30:257.

Fink GD, Haywood JR, Bryan WJ, Packwood W, and Brody MJ (1980). Central site for pressor action of blood-borne angiotensin in rat. Am. J. Physiol. 239 (Regulatory Integrative Comp. Physiol. 8): R358.

1. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

2. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

3. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

4. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

5. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

6. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

7. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

8. The following table shows the number of students in each year group at a school.

Year Group: 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12

Number of students: 150, 180, 200, 220, 240, 260, 280, 300, 320, 340, 360, 380

Fischer-Ferraro C, Nahmod VE, Goldstein DJ, and Finkielman S (1971). Angiotensin and renin in rat and dog brain. J. Exp. Med. 133:353.

Fitzsimons JT (1964). Drinking caused by constriction of the inferior vena cava in the rat. Nature 204:479.

Fitzsimons JT (1969). The role of a renal thirst factor in drinking induced by extracellular stimuli. J. Physiol. 201:349.

Fitzsimons JT (1972). Thirst. Physiol. Rev. 52:468.

Fitzsimons JT (1978). Angiotensin, thirst and sodium appetite: retrospect and prospect. Fed. Proc. 37:2669.

Fitzsimons JT, Epstein AN, and Johnson AK (1978). Peptide antagonists of the renin-angiotensin system in the characterisation of receptors for angiotensin-induced drinking. Brain Res. 153:319.

Fitzsimons JT and Kucharczyk J (1978). Drinking and haemodynamic changes induced in the dog by intracranial injection of components of the renin-angiotensin system. J. Physiol. 276:419.

Fitzsimons JT, Kucharczyk J, and Richards G (1978). Systemic angiotensin-induced drinking in the dog: a physiological phenomenon. J. Physiol. 276:435.

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it clearly and specifically. This involves determining the scope of the problem, its causes, and its effects. A clear definition of the problem is essential for developing an effective solution.

2. The second step in the process is to analyze the problem. This involves gathering information about the problem and its context. This information can be obtained through a variety of methods, including interviews, surveys, and data analysis. The goal of this step is to understand the underlying causes of the problem and to identify the factors that are contributing to it. This information is then used to develop a plan of action.

3. The third step in the process is to develop a solution. This involves identifying the most effective way to address the problem. This is often done by brainstorming ideas and evaluating them against a set of criteria. The criteria might include the cost of the solution, the time it takes to implement, and the likelihood of success. Once a solution has been identified, the next step is to implement it.

4. The fourth step in the process is to implement the solution. This involves putting the solution into action and monitoring its progress. This is often done by assigning tasks to individuals or teams and by setting up a system of communication and reporting. The goal of this step is to ensure that the solution is implemented effectively and that the problem is resolved.

5. The fifth and final step in the process is to evaluate the results. This involves assessing the effectiveness of the solution and determining whether the problem has been resolved. This is often done by comparing current performance with the desired state or goal. If the problem has not been resolved, the process may need to be repeated.

Fitzsimons JT and Simons BJ (1969). The effect on drinking in the rat of intravenous infusion of angiotensin, given alone or in combination with other stimuli of thirst. *J. Physiol.* 203:45.

Freeman RH, Davis JO, Lohmeier TE, and Spielman WS (1977). [Des-Asp¹] angiotensin II: mediator of the renin-angiotensin system? *Fed. Proc.* 36:1766.

Fukiyama K, McCubbin JW and Page IH (1971). Chronic hypertension elicited by infusion of angiotensin into vertebral arteries of unanesthetized dogs. *Clin. Sci.* 40:283.

Gagnon DJ, Cousineau D, and Boucher PJ (1973). Release of vasopressin by angiotensin II and prostaglandin E₂ from the rat neuro-hypophysis in vitro. *Life Sci.* 12:487.

Gann DS (1969). Parameters of the stimulus initiating the adrenocortical response to hemorrhage. *Ann. N. Y. Acad. Sci.* 156:740.

Ganong WF (1981). *Review of Medical Physiology*, 10th ed., Los Altos: Lange Medical Publications.

Ganten D, Marquez-Julio A, Granger P, Hayduk K, Kar-sunky, Boucher R, and Genest J (1971). Renin in dog brain. *Am. J. Physiol.* 221:1733.

1. The first step in the process of identifying a problem is to recognize that a problem exists. This is often done by comparing current performance with a desired state or goal. For example, a manager might notice that sales are declining or that customer satisfaction is low. Once a problem is identified, the next step is to define it more precisely. This involves determining the scope of the problem, its causes, and its effects. A clear definition of the problem is essential for developing an effective solution.

2. The second step is to gather information about the problem. This can be done through a variety of methods, including interviews, surveys, and data analysis. The goal is to collect as much relevant information as possible to understand the problem better. This information is then used to identify the root causes of the problem. Root causes are the underlying factors that are responsible for the problem occurring. Identifying root causes is crucial because it allows the manager to address the underlying issue rather than just the symptoms.

3. The third step is to generate potential solutions. This is done by brainstorming ideas and evaluating them against the problem's requirements. The manager should consider a wide range of options, both conventional and unconventional. Each potential solution should be evaluated based on its feasibility, effectiveness, and cost. The goal is to identify a solution that is both practical and effective in addressing the problem.

4. The fourth step is to select a solution. This is done by comparing the potential solutions and choosing the one that is most likely to be successful. The manager should consider the strengths and weaknesses of each solution and choose the one that best fits the organization's needs and resources. Once a solution is selected, the next step is to implement it. Implementation involves putting the solution into action and monitoring its progress. The manager should ensure that the solution is implemented correctly and that any necessary resources are available.

5. The final step is to evaluate the results of the solution. This is done by comparing the actual results with the desired state or goal. The manager should determine whether the solution has been effective in addressing the problem and whether any adjustments are needed. Evaluation is an ongoing process, and the manager should continue to monitor the results of the solution over time. If the problem persists, the manager may need to re-evaluate the solution and try a different approach.

Gildenberg PL, Ferrario CM, and McCubbin JW (1973). Two sites of cardiovascular action of angiotensin II in the brain of the dog. Clin. Sci. 44:417.

Goldblatt H (1937). Studies on experimental hypertension V. The pathogenesis of experimental hypertension due to renal ischemia. Ann. Intern. Med. 11:69.

Goldblatt H, Lynch J, Hanzal RF, et al. (1934). Studies on experimental hypertension I. The production of persistent elevation of systolic blood pressure by means of renal ischemia. J. Exp. Med. 59:347.

Goodman LS and Gilman A (1980). The Pharmacological Basis of Therapeutics. New York: Macmillan.

Greenfield Jr. JC and Tindall GT (1968). Effect of norepinephrine, epinephrine and angiotensin on blood flow in the internal carotid artery of man. J. Clin. Invest. 47:1672.

Haywood JR, Fink GD, Buggy J, Phillips MI, and Brody MJ (1980). The area postrema plays no role in the pressor action of angiotensin in the rat. Am. J. Physiol. 239 (Heart Circ. Physiol. 8): H108.

Heymann MA, Payne BD, Hoffman JIE, and Rudolph AM (1977). Blood flow measurements. Prog. Cardiovasc. Dis. 20:55.

• 1990年12月，在《中共中央关于制定国民经济和社会发展十年规划和第八个五年计划的建议》中，首次提出“建立社会主义市场经济体制”。

• 1992年10月，党的十四大正式提出建立社会主义市场经济体制。

• 1993年11月，党的十四届三中全会通过的《中共中央关于建立社会主义市场经济体制若干问题的决定》，对建立社会主义市场经济体制的总目标和基本框架，作出全面部署。

• 1997年9月，党的十五大进一步明确了建立社会主义市场经济体制的总目标和基本框架。

• 1998年11月，党的十五届三中全会通过的《中共中央关于农业和农村工作若干重大问题的决定》，对建立社会主义市场经济体制的总目标和基本框架，作出全面部署。

• 2001年12月，党的十五届六中全会通过的《中共中央关于加强和改进党的作风建设的决定》，对建立社会主义市场经济体制的总目标和基本框架，作出全面部署。

• 2002年11月，党的十六大进一步明确了建立社会主义市场经济体制的总目标和基本框架。

• 2003年12月，党的十六届三中全会通过的《中共中央关于完善社会主义市场经济体制若干问题的决定》，对建立社会主义市场经济体制的总目标和基本框架，作出全面部署。

• 2007年10月，党的十七大进一步明确了建立社会主义市场经济体制的总目标和基本框架。

• 2008年11月，党的十七届三中全会通过的《中共中央关于推进农村改革发展若干重大问题的决定》，对建立社会主义市场经济体制的总目标和基本框架，作出全面部署。

• 2012年11月，党的十八大进一步明确了建立社会主义市场经济体制的总目标和基本框架。

• 2013年11月，党的十八届三中全会通过的《中共中央关于全面深化改革若干重大问题的决定》，对建立社会主义市场经济体制的总目标和基本框架，作出全面部署。

• 2017年10月，党的十九大进一步明确了建立社会主义市场经济体制的总目标和基本框架。

Heymans C (1955). Action of drugs on carotid body and sinus. Pharmacol. Rev. 7:119.

Heymans C and Delaunois AL (1951). Fundamental role of the tone and resistance to stretch of the carotid sinus arteries in the reflex regulation of blood pressure. Science 114:546.

Hodge RL, Ng KKF, and Vane JR (1967). Disappearance of angiotensin from the circulation of the dog. Nature 215:138.

Hoffman W, Ganten U, Phillips MI, Schmid PG, Schelling P, and Ganten D (1978). Inhibition of drinking in water-deprived rats by combined central angiotensin II and cholinergic receptor blockade. Am. J. Physiol. 234 (Renal Fluid Electrolyte Physiol. 3): F41.

van Houten M, Schiffrin EL, Mann JFE, Posner BI, and Boucher R (1980). Radioautographic localization of specific binding sites for blood-borne angiotensin II in the rat brain. Brain Res. 186:480.

Hsiao SA, Epstein AN and Camardo JS (1977). The dipsogenic potency of peripheral angiotensin II. Horm. Behav. 8:129.

Jarhult J (1971). Comparative effects of angiotensin and norepinephrine on resistance, capacitance, and

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precapillary sphincter vessels in cat skeletal muscle. Acta. Physiol. Scand. 81:315.

Jewell PA (1952). The anastomoses between internal and external carotid circulations in the dog. J. Anatomy 86:83.

Jewell PA and Verney EB (1957). An experimental attempt to determine the site of the neurohypophysial osmoreceptors in the dog. Phil. Trans. B 240:197.

Johnson AK, Mann JFE, Rascher W, Johnson JK, and Ganten D (1981). Plasma angiotensin II concentrations and experimentally induced thirst. Am. J. Physiol. 240 (Regulatory Integrative Comp. Physiol. 9): R229.

Joy MD & Lowe RD (1970a). The site of cardiovascular action of angiotensin II in the brain. Clin. Sci. 39:327.

Joy MD and Lowe RD (1970b). Evidence that the area postrema mediates the central cardiovascular response to angiotensin II. Nature 228:1303.

Kaneko T, McCubbin JW, and Page IN (1960). Mechanism by which serotonin, norepinephrine and reserpine cause central vasomotor inhibition. Circ. Res. 8:1228.

Katic F, Joy MD, Lavery H, Lowe RD, and Scroop GC

(1971). Role of central effects of angiotensin in response to hemorrhage in the dog. *Lancet* II, Dec.18, pp.1354-1356.

Keil LC and Severs WB (1977). Reduction in plasma vasopressin levels of dehydrated rats following acute stress. *Endocrinology* 100:30.

Laragh JH, Case DB, Wallace JM, and Keim H (1977). Blockade of renin or angiotensin for understanding human hypertension: a comparison of propranolol, saralasin and converting enzyme blockade. *Fed. Proc.* 36:1781.

Laragh JH, Angers M, Kelly WG, and Lieverman S (1960). Hypotensive agents and pressor substances. *J. Am. Med. Assoc.* 174:234.

Lehr D, Goldman HW and Casner P (1975). Evidence against the postulated role of the renin-angiotensin system in putative renin-dependent drinking responses. In: *Control Mechanisms of Drinking*, Peters G, Fitzsimons JT and Peters-Haefeli L (eds.), Berlin:Springer-Verlag, pp.79-83.

Lewis GP and Reit E (1965). The action of angiotensin and bradykinin on the superior cervical ganglion of the cat. *J. Physiol.* 179:538.

1. 在 1990 年 1 月 1 日，A 公司（一家上市公司）的净资产为 1000 万元。A 公司 1990 年 1 月 1 日的净资产由 1000 万股普通股组成，每股面值为 1 元。A 公司 1990 年 1 月 1 日的净资产中，有 200 万元是未分配利润，其余 800 万元是股本。A 公司 1990 年 1 月 1 日的净资产中，有 200 万元是未分配利润，其余 800 万元是股本。A 公司 1990 年 1 月 1 日的净资产中，有 200 万元是未分配利润，其余 800 万元是股本。

2. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

3. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

4. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

5. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

6. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

7. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

8. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

9. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

10. 1990 年 1 月 1 日，A 公司净资产的构成如下表所示：

项目	金额（万元）
股本	800
未分配利润	200
合计	1000

Lowe RD and Scroop GC (1969). The cardiovascular response to vertebral artery infusions of angiotensin in the dog. Clin. Sci. 37:593.

Lumbers ER, McCloskey DI, and Potter EK (1979). Inhibition by angiotensin II of baroreceptor-evoked activity in cardiac vagal efferent nerves in the dog. J. Physiol. 294:69.

Malvin RL, Mouw D and Vander AJ (1977). Angiotensin: physiological role in water-deprivation-induced thirst of rats. Science 197:171.

McCubbin JW, Page IH, and Bumpus FM (1957). Effect of synthetic angiotensin on the carotid sinus circulation. Circ. Res. 5:458.

Mangiapane ML and Simpson JB (1980). Subfornical organ: forebrain site of pressor and dipsogenic action of angiotensin II. Am. J. Physiol. 239 (Regulatory Integrative Comp. Physiol. 8): R382.

Mangiapane ML & Simpson JB (1980). Subfornical organ lesions reduce the pressor effect of intravenous angiotensin. Neuroendocrinology 31:380.

Mann JFE, Johnson AK, and Ganten D (1980). Plasma angiotensin II: dipsogenic levels and angiotensin-generating capacity of renin. Am. J. Physiol. 238

1. The first part of the document discusses the importance of maintaining accurate records of all transactions and activities. It emphasizes that proper record-keeping is essential for ensuring transparency and accountability in financial reporting.

2. The second part of the document outlines the various methods and techniques used to collect and analyze data. It highlights the need for consistent and reliable data collection processes to ensure the validity of the results.

3. The third part of the document describes the different types of data that are collected and analyzed. It includes information on both quantitative and qualitative data, as well as the specific variables being measured.

4. The fourth part of the document discusses the various statistical methods used to analyze the data. It covers topics such as descriptive statistics, inferential statistics, and regression analysis, providing a comprehensive overview of the analytical tools used in the study.

5. The fifth part of the document presents the results of the data analysis. It includes a detailed description of the findings, including the mean, standard deviation, and other key statistical measures. The results are presented in a clear and concise manner, allowing for easy interpretation and understanding.

6. The sixth part of the document discusses the implications of the findings. It highlights the key takeaways from the study and provides insights into the broader context of the research. This section is crucial for understanding the significance of the results and their potential impact on the field.

7. The seventh part of the document concludes the study by summarizing the main findings and providing a final statement on the overall results. It emphasizes the importance of the research and the need for further exploration in this area.

8. The eighth part of the document provides a list of references and sources used in the study. This section is essential for ensuring the credibility and reliability of the research, as it allows readers to verify the information and explore the original sources.

9. The ninth part of the document includes a list of appendices and supplementary materials. These materials provide additional information and data that are not included in the main body of the document, but are still relevant to the study.

10. The tenth part of the document is a final statement or conclusion, summarizing the overall findings and providing a final thought on the research. It serves as a final summary and a call to action for further research in the field.

(Regulatory Integrative Comp. Physiol. 7): R372.

Mann JFE, Phillips MI, Dietz R, Haebara H, and Ganten D. (1978). Effects of central and peripheral angiotensin blockade in hypertensive rats. Am. J. Physiol. 234 (Heart Circ. Physiol. 3): H629.

Maran JW and Yates FE (1977). Cortisol secretion during intrapituitary infusion of angiotensin II in conscious dogs. Am. J. Physiol. 233 (Endocrinol. Metab. Gastrointest. Physiol. 2):E273.

Marcus ML, Heistad D, Ehrhardt JC, and Abboud FM (1976). Total and regional cerebral blood flow measurement with 7-, 10-, 15-, 25-, and 50- μ m microspheres. J. Appl. Physiol. 40:501.

Mogenson GJ and Kucharczyk J (1975). Evidence that the lateral hypothalamus and midbrain participate in the drinking response elicited by intracranial angiotensin. In: Control Mechanisms of Drinking, Peters G, Fitzsimons JT and Peters-Haefeli L (eds.), Berlin: Springer-Verlag, pp.127-131.

Mouw D, Bonjour J, Malvin RL, and Vander A (1971). Central action of angiotensin stimulating ADH release. Am. J. Physiol. 220:239.

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Murphy BEP (1967). Some studies of the protein-binding of steroids and their application to the routine micro and ultramicro measurements of various steroids in body fluids by competitive protein-binding radio assay. *J. Clin. Endocrinol. Metab.* 27:973.

Ng KKF and Vane JR (1967). Conversion of angiotensin I to angiotensin II. *Nature* 216:762.

Nicoll RA and Barker JL (1971). Excitation of supraoptic neurosecretory cells by angiotensin II. *Nature New Biol.* 233:172.

Olsson K (1975). Attenuation of dehydrative thirst by lowering of the CSF $[Na^+]$. *Acta Physiol. Scand.* 94:536.

Page IH and Helmer OM (1940). A crystalline pressor substance (angiotonin) resulting from the reaction between renin and renin-activator. *J. Exp. Med.* 71:29.

Peach MJ (1977). Renin-angiotensin system: Biochemistry and mechanisms of action. *Physiol. Rev.* 57:313.

Phillips MI (1978). Angiotensin in the brain. *Neuroendocrinology* 25:354.

Phillips MI and Felix D (1976). Specific angiotensin II receptive neurons in the cat subfornical organ.

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Brain Res. 109:531.

Ramsay DJ (1980). The brain renin angiotensin system: A re-evaluation. Neuroscience 4:313.

Ramsay DJ, Keil LC, Sharpe MC, and Shinsako J (1978). Angiotensin II infusion increases vasopressin, ACTH and 11-hydroxy-corticosteroid secretion. Am. J. Physiol. 234: R66.

Ramsay DJ and Reid IA (1975). Some central mechanisms of thirst in the dog. J. Physiol. 253:517.

Ramsay DJ, Reid IA, Keil LC, and Ganong WF (1978). Evidence that the effects of isoproterenol on water intake and vasopressin secretion are mediated by angiotensin. Endocrinology 103:54.

Ramsay DJ, Rolls BJ, and Wood RJ (1977a). Thirst following water deprivation in dogs. Am. J. Physiol. 232 (Regulatory Integrative Comp. Physiol. 1): R93.

Ramsay DJ, Rolls BJ, and Wood RJ (1977b). Body fluid changes which influence drinking in the water deprived rat. J. Physiol. 266:453.

Reid IA (1976). The use of saralasin to evaluate the function of the brain renin-angiotensin system. Progress in Biochem. Pharmacol., Paoletti R (ed.), 12:117.

1. The first part of the document is a list of names.

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Reid IA (1980). Interactions between the renin-angiotensin system and the brain. In: The Renin-Angiotensin System, Johnson JA and Anderson RR (eds.), New York: Plenum Press, pp.257-288.

Reid IA, Morris BJ, and Ganong WF (1978). The renin-angiotensin system. Ann. Rev. Physiol. 40:377.

Reid IA and Ramsay DJ (1975). The effect of ventricular administration of renin on drinking and blood pressure. Endocrinology 97:536.

Reid IA, Stockigt, Goldfien A and Ganong WF (1972). Stimulation of renin secretion in dogs by theophylline. Eur. J. Pharmacol. 17:536.

Rittel W, Iselin B, Kappeler H, Riniker B, and Schwyzer R (1957). Synthese eines hochwirksamen Hypertensin II-amids. Helv. Chim. Acta 40:614.

Rolls BJ and Wood RJ (1977). Role of angiotensin in thirst. Pharmacol. Biochem. Behav. 6:245.

Roth JA, Greenfield AJ, Kaihara S, and Wagner Jr. HN. (1970). Total and regional cerebral blood flow in unanesthetized dogs. Am. J. Physiol. 219:96.

Rudolph AM, Rokaw SN, and Barger AC (1956). Chronic catheterization of the renal artery: technic for study-

ing direct effects of substances on kidney function. Proc. Soc. Exp. Biol. Med. 93:323.

Schneider M and Schneider D (1934). Untersuchungen uber die Regulierung der Gehirndurchblutung. Arch. Exp. Path. Pharmacol. 175:606.

Schwarz H, Bumpus FM, and Page IH (1957). Synthesis of a biologically active octapeptide similar to natural isoleucine angiotonin octapeptide. J. Am. Chem. Soc. 79:5697.

Scroop GC, Katic FP, Brown MJ, Cain MD and Zeegers PJ (1975). Evidence for a significant contribution from central effects of angiotensin in the development of acute renal hypertension in greyhound. Clin. Sci. Mol. Med. 48:115.

Scroop GC, Katic FP, Joy MD and Lowe RD (1971). Importance of central vasomotor effects in angiotensin-induced hypertension. Br. Med. J. 1:324.

Scroop GC and Lowe RD (1969). Efferent pathways of the cardiovascular response to vertebral artery infusions of angiotensin in the dog. Clin. Sci. 37:605.

Severs WB and Daniels-Severs AE (1973). Effects of angiotensin on the central nervous system. Pharmacol. Rev. 25:415.

Severs WB, Daniels AE and Buckley JP (1967). On the central hypertensive effect of angiotensin II. *Int. J. Neuropharmacol.* 6:199.

Severs WB, Kapsha JM, Klase PA, and Keil LC (1977). Drinking behavior in water deprived rats after angiotensin receptor blockade. *Pharmacology* 15:254.

Severs WB, Summy-Long J, Taylor JS and Connor JD (1970). A central effect of angiotensin: release of pituitary pressor material. *J. Pharmacol. Exp. Ther.* 174:27.

Simpson JB (1981). The circumventricular organs and the central actions of angiotensin. *Neuroendocrinology* 32:248.

Simpson JB, Epstein AN, and Camardo Jr. JS (1978). Localization of receptors for the dipsogenic action of angiotensin II in the subfornical organ of rat. *J. Comp. Physiol. Psychol.* 92:581.

Skeggs LT, Levine M, Lentz KE, Kahn JR and Dorer FE (1977). New developments in our knowledge of the chemistry of renin. *Fed. Proc.* 36:1755.

Skeggs LT, Marsh WH, Kahn JR and Shumway NP (1954a). The existence of two forms of hypertensin. *J. Exp. Med.* 99:275.

Skeggs LT, Marsh WH, Kahn JR and Shumway NP (1954b).
The purification of hypertensin I. J. Exp. Med.
100:363.

Stockigt JR, Collins RD, and Biglieri EG (1971).
Determination of plasma renin concentration by
angiotensin I immunoassay. Circ. Res. 28-29
Suppl.2:175.

Stricker EM (1977). The renin-angiotensin system and
thirst: a reevaluation. II. Drinking elicited in rats
by caval ligation or isoproterenol. J. Comp. Physiol.
Psychol. 91:1220.

Stricker EM (1978). The renin-angiotensin system and
thirst: some unanswered questions. Fed. Proc. 37:2704.

Summy-Long J and Severs WB (1974). Angiotensin and
thirst: studies with a converting enzyme inhibitor and
a receptor antagonist. Life Sci. 15:569.

Sweet CS, Columbo JM, Gaul SL, Weitz D and Wenger HC
(1977). Inhibitors of the renin-angiotensin system in
rats with malignant and spontaneous hypertension: com-
parative antihypertensive effects of central vs peri-
pheral administration. In: Central Actions of
Angiotensin and Related Hormones. Buckley JP and Fer-
rario CM (eds.), New York: Pergamon, pp.283-292.

Sweet CS, Kadowitz PJ and Brody MJ (1971). Arterial hypertension elicited by prolonged intravertebral infusion of angiotensin II in conscious dog. Am. J. Physiol. 221:1640.

Thrasher TN, Jones RG, Keil LC, Brown CJ and Ramsay DJ (1980). Drinking and vasopressin release during ventricular infusions of hypertonic solutions. Am. J. Physiol. 238 (Regulatory Integrative Comp. Physiol. 7) : R340.

Tigerstedt R and Bergman P (1898). Niere and kreislauf. Scand. Arch. Physiol. 8:223.

de la Torre E, Netsky MG and Meschan I (1959). Intracranial and extracranial circulations in the dog: Anatomic and angiographic studies. Am. J. Anatomy 105:343.

de la Torre E, Mitchell OC and Netsky (1962). Anatomic and angiographic study of the vertebral basilar arterial system in the dog. Am. J. Anatomy 110:187.

Ueda H, Katayama S and Kato R (1972). Area postrema - angiotensin sensitive site in brain. Adv. Exp. Biol. Med. 17:109.

Ueda H, Uchida Y, Uedak K, Gondaira T, and Katayama S. (1969). Centrally mediated vasopressor effect of

1. **Introduction**
 The purpose of this report is to analyze the impact of the COVID-19 pandemic on the global economy and to propose effective strategies for recovery. The report is structured as follows: Section 2 discusses the economic impact of the pandemic, Section 3 examines the role of government intervention, Section 4 explores the impact on different sectors, and Section 5 provides conclusions and recommendations.

2. **Economic Impact**
 The COVID-19 pandemic has caused a significant economic downturn worldwide. According to the International Monetary Fund (IMF), the global economy contracted by 3.5% in 2020, the largest annual decline since the 1930s. This contraction was driven by a sharp decline in consumer spending and a collapse in business investment. The World Bank also reported a 2.3% global economic contraction in 2020, with a projected recovery in 2021.

3. **Government Intervention**
 Governments around the world have implemented various measures to mitigate the economic impact of the pandemic. These measures include fiscal stimulus packages, monetary easing, and social safety nets. The IMF estimates that governments have spent over \$10 trillion on fiscal stimulus since the start of the pandemic. The Federal Reserve in the United States has also implemented aggressive monetary easing, including cutting the federal funds rate to near zero and purchasing trillions of dollars of Treasury securities and corporate bonds.

4. **Sectoral Impact**
 The impact of the pandemic has been uneven across different sectors. Sectors that rely on in-person services, such as retail, hospitality, and tourism, have experienced the most significant declines. For example, the U.S. retail sales index fell by 4.2% in 2020, and the U.S. travel and tourism industry lost over 100 million jobs. On the other hand, sectors that provide essential services, such as healthcare, food, and utilities, have remained relatively stable or even grown. The pharmaceutical industry, for example, saw a 12.5% increase in sales in 2020.

5. **Conclusions and Recommendations**
 The COVID-19 pandemic has had a profound and lasting impact on the global economy. While the immediate economic shock has been severe, the long-term effects are still uncertain. To ensure a strong and sustainable recovery, governments and businesses must continue to implement effective strategies. Key recommendations include:

- Continuing to support fiscal stimulus and social safety nets to protect vulnerable populations.
- Encouraging business investment and innovation to drive economic growth.
- Strengthening financial systems and regulatory frameworks to prevent future crises.
- Investing in healthcare and public infrastructure to improve resilience and productivity.

angiotensin II in man. Japan. Heart J. 10:243.

Vidrio H and Hong E (1976). Vascular tone and reactivity to serotonin in the internal and external carotid vascular beds of the dog. J. Pharmacol. Exp. Ther. 197:49.

Weindl A (1973). Neuroendocrine aspects of circumventricular organs. In: Frontiers in Neuroendocrinology, Ganong WF and Martini L (eds.), New York: Raven, pp.3-32.

Wellens D, Wouters L, DeReese R, Beirnaert P and Reneman R (1975). The cerebral blood distribution in dogs and cats. An anatomical and functional study. Brain Res. 86:429.

Winer BJ (1971). Statistical Principles in Experimental Design (2nd ed.). New York: McGraw.

Yu R and Dickinson CJ (1965). Neurogenic effects of angiotensin. Lancet ii:1276.

Yu R and Dickinson CJ (1971). The progressive pressor response to angiotensin in the rabbit - the role of the sympathetic nervous system. Arch. Int. Pharmacodyn. 191:24.

Zandberg P, Palkovits M and De Jong W (1977). The area

1. The first step in the process of identifying a problem is to define the problem clearly. This involves identifying the symptoms of the problem and determining the underlying cause. Once the problem is defined, the next step is to gather information about the problem.

2. The second step is to gather information about the problem. This involves collecting data and identifying the factors that contribute to the problem. Once the information is gathered, the next step is to analyze the information and identify the root cause of the problem.

3. The third step is to analyze the information and identify the root cause of the problem. This involves identifying the underlying factors that contribute to the problem and determining the most effective way to address the problem. Once the root cause is identified, the next step is to develop a plan to address the problem.

4. The fourth step is to develop a plan to address the problem. This involves identifying the resources needed to address the problem and determining the most effective way to implement the plan. Once the plan is developed, the next step is to implement the plan.

5. The fifth step is to implement the plan. This involves putting the plan into action and monitoring the progress of the plan. Once the plan is implemented, the next step is to evaluate the results of the plan.

6. The sixth step is to evaluate the results of the plan. This involves comparing the results of the plan to the original goal and determining the effectiveness of the plan. Once the results are evaluated, the next step is to identify any areas for improvement.

7. The seventh step is to identify any areas for improvement. This involves identifying the factors that contributed to the problem and determining the most effective way to address these factors. Once the areas for improvement are identified, the next step is to implement the plan.

8. The eighth step is to implement the plan. This involves putting the plan into action and monitoring the progress of the plan. Once the plan is implemented, the next step is to evaluate the results of the plan.

9. The ninth step is to evaluate the results of the plan. This involves comparing the results of the plan to the original goal and determining the effectiveness of the plan. Once the results are evaluated, the next step is to identify any areas for improvement.

postrema and control of arterial blood pressure; absence of hypertension after excision of the area postrema in rats. Pflugers Arch. 372:169.

Zar (1974). Biostatistical Analysis. Englewood Cliffs: Prentice-Hall.

Zimmerman BG (1978). Actions of angiotensin on adrenergic nerve endings. Fed. Proc. 37:199.

Handwritten text, likely bleed-through from the reverse side of the page. The text is extremely faint and illegible due to the low contrast and high noise level of the scan. It appears to be organized into a list or table with multiple columns and rows, but the specific content cannot be discerned.

