CHAPTER XI

OBSERVATIONS IN ANIMAL DISEASES

DAIRY CATTLE PLUS PUS INFECTIONS

We are indebted to Dr. David Arnott for development of the use of the carbonyl catalysts in the diseases of Diary Cattle. The enormous amount of data meticulously built up by the scientists of the Ministry of Agriculture and the University of British Columbia, Canada showed the basic place of this therapy in the tissue oxidation processes that use sugar. Thus some 95 to 100% of cases of acetonemia are quickly cured by a single injection per animal in both the acute and the chronic cases. The hundreds of cases treated for infectious mastitis show a rapid cure in the acute cases with hemolytic streptococcus and staphylococcus cases approaching 90% while the chronic cases. with much fibrosis showed something like an 80% cure rate with restoration of function, and replacement of the fibrosis with normal gland tissue. This is the only therapy that has ever demonstrated this result. In Brazil we showed that the fibrosis that completely invaded the udder and closed the teats after local treatments with various antibiotics, could be completely cured by this therapy too. The fibrosis and the infection it contained were eliminated by replacement with normal functioning gland tissue.

However the reports of the Minister of Agriculture of British Columbia to the Parliament in the official bulletins of years 1944 through 1949 inclusive not only verify our working hypothesis, in general, but also supply bacteriological counts before and after treatment which indicate the pathogenic germs of highest virulance before treatment may rapidly drop in numbers a few days after treatment when the udders are undergoing healing and also that where the injury was very severe, the bacteria at times increased in number during healing, and while the toxicity of the animal was rapidly disappearing. Our interpretation of this oft observed affair, especially in gangre-

nous mastitis, is that the germ became no longer toxic, as we see in huge tuberculous cavitations in man, and indeed appear to help in the clean-up process. As soon as the tissue debris is eliminated, they rapidly disappear, even before the cavity or lesion is healed by tissue reconstruction. They thus appear to have shared in the benefits from the carbonyl catalysts and become normal useful members of the biological economy, and help clean up the mess they formerly caused.

It was Dr. Arnott's contribution to the court defense of our work that went a long way toward preserving this therapy for humanity. The United States government claimed that the cases we cured, never were sick, and if they were, they did not get well, and if they got well, some other therapy cured them, and if no other therapy was used, they were cured by psychology. The cure of dairy cattle of infections and metabolic disturbances from which no other treatment could free them, would have had to be done by psychology if our treatment did not do it, and as the experts from British Columbia under Dr, Arnott's leadership demonstrated, the cures could only have come from our treatment, as the government witnesses had to admit, psychology could not enter the picture at all.

BRUCELLOSIS

It was the cure of dairy farmers suffering with Brucellosis that gave start to the treatment of this disease in cattle in Canada by Dr. Arnott. The cure percentage ran somewhat over 80%, in dairy cows, and this is what was recorded in the Michigan experiments, as well as those conducted in a small number of cows in Brazil. In the latter cases which I treated for the Ministry of Agriculture the cases were far advanced and of the broken down type. One had a severe infection of the udder with the diphtheria bacillus which completely involved three quarters and half of the other quarter. It was resistant to all forms of treatment and pronounced entirely hopeless by the University Pathologist who had supervised this case. They were all cachectic with "moth-eaten" fur, or with an arthritis, ulceration, infertility or some other complication. Of five such cases four gave birth to normal calves at normal term, and the placenta in each case was found to be structurally and bacterio-

logically normal, and free from the Brucella germ. The other case aborted within three months of receiving the treatment, but no follow-up was had to determine if the cure came after the third month which is usually the case. The cow with both brucellosis and Corynebacterium mastitis was fully cured of both infections—a surprise to all observers. She gave birth to a normal calf and the placenta was proven normal and free from infection. No Brucella germs were found, and the udder normalized completely without fibrosis, with return of full lactation.

Absorption of the fetus no longer occurred after the treatment and thus the normal reproductive physiology was restored whether the interference was a matter of dietary insufficiency, or from selenium in the plants, soil, or water, or if the injury came from the Brucellosis germ. High potency oxidation catalysis removed the interference and restored tissue function, energy production so normal behaviour could be resumed. The subject of infertility in cattle is treated by Dr. Bruce Richardson in his graduation thesis from the University of British Columbia, and by Dr. Wood the professor of Pathology. Their recovery percentages were about 72% while those in Michigan ran much higher. Thus the environmental features deserve consideration, and these are vastly different in the two places.

Infertility in cattle as in man may have a complex origin and many factors may be determinative. Thus imperfection in food, toxins of various origins, and those as selenium coming from the soil are definite causes. Yet the poison of Brucellosis is most important. Correction of the feeding may be somewhat helpful, but in the confirmed cases a basic boost to the metabolism able to burn the hindering toxin, out of the way is needed.

VIRUS INFECTIONS IN ANIMALS

RABIES[1]

Lytic Integration As In Diagram V, (a)

The Virus

There are three varieties of Rabies virus at present, the original "Street" variety, and the two adapted types that are grown on chick embryos and that are fixed to nerve tissue

culture by brain innoculations. Both adapted types tend to revert back to the parent "Street" type under conditions that offer structural units possessed by the latter, but lost in the cultural adaptations to only brain and chick embryo vegetation. Virulence is largely lost by the two types of adaptation; and this fact forms the basis that encourages the production of the Fleury type live virus vaccine.

Immunity to virus diseases has not responded well to the killed virus vaccines, and as it is recognized that the only immunity of value to virus caused disease follows the recovery from the active infection, the effort to produce the disease in a mild readily vanquished form seems logical. However, the most diligent efforts to master the details that must be encountered have shown that real success has not been attained as yet, and as we are combating the most basic laws of Nature, the hope for success is not too promising either. Cow pox is a natural live virus vaccine against Small Pox. Yet it must be an attenuated form of the latter, since under favorable conditions it becomes a most virulant killer small pox virus.

This has happened under my observation with Aftosa vaccine several times, and could be the cause of the scandal with the "Polio" vaccines. Practical experience convinces one that mild or defective virus of "Polio," Small Pox, and other diseases, infects the public as widespread as our Type III integration with host cells on a large scale, and that such infection for example gives the serological reactions for the "Polio" sensitivity or "immunity" that is diagnosed as Infantile Paralysis when the serological tests are given those with the "flu" or an intestinal indigestion, and classified as non-paralytic Poliomyelitis. However, an innocent Polio vaccine may supply the units which when added to those of the silent infection (neither one of which could supply the required units alone for a provirus that could mature to engage in actual vegetation), would together make up the material required for a parent killer type, and thus precipitate a highly fatal epidemic in any large city. This is true because so many people without "Polio" are serologically sensitive, and are protected from the virulent infection coming

[1]This is a report on the first cases of Rabies ever cured. The report has been delayed one year to make sure of the permanency.

from without by this natural vaccine type infection. If it is let alone it protects, but when one tries to improve upon it by any type of virus vaccination, the disaster can be created within the system of the victim. What applies to one person applies to most of the community. Many virus infections including cancer may be represented by this Type III silent integration.

However, in Rabies the situation is quite different, and if not vaccinated previously no such catastrophe would be expected. The epidemic to be described therefore must be accounted for by the presence of active live, fully virulent Rabies virus in the vaccine, since it attacked calves that are not exposed to any type of Rabies virus at all, and hence have nothing to add to the vaccine to make it malignant.

There is another consideration that deserves thought, namely, that the epidemic we will describe took place pre-eminently in bull calves several months old. Here the activated amines and amidines produced by the testis may possibly offer the attenuated vaccine virus the amine groups it needs to make pathogenic azomethine bondings with the host cell, and which amine groups might conceivably be taken up into the virus structure as happens when an excess of an amino acid supplied to the host culture, is built into the virus's protein in such proportions as to change its serology in a transmissable way. If such a thing happens, bull calves would tend to change an innocent attenuated virus to a killer type. Still females were also affected. So this explanation is weak and can be discarded for a more practical one that follows.

In this epidemic short needles were used to make the injections of the vaccine. Since the Zebu possesses a skin structure with a non-elastic peniculus that could hold the injected material a long time and prevent its absorption, the short needle would only deliver the material into the muscle in the calves and not into adult animals. Such protection may account for the unusual resistance of the Zebu to snake-bite poisoning. In the present epidemic it probably protected the larger animals. Our suspicion is that the vaccine contained virulent Rabies virus that had taken advantage of some encouragement to revert back to the parent un-attenuated type, before it was injected.

One must remember the tendency to revert to the parent type is not lost even by prolonged passage through attenuating culture as it is a basic protective response.

The Integrated State

The briefest review of virus parasitism demonstrates a common pattern of action to which the facts we have to contribute here give confirmation. It is now agreed without exception that once a pathogenic virus has penetrated its host cell and become integrated therewith, that is, about a minute and a half after penetration, no amount of immune serum or vaccine can rescue the host cell from destruction. Evidently the union between the host cell and the integrated virus is so very intimate and fixed that displacement by other combining substances is entirely out of the question. Immune sera only partially neutralize the virus anyway, and the latter is easily separated therefrom. The host cell-virus bonding is much stronger. That is why we found it necessary to work out a system of oxidizing the virus off at the azomethine bonding so as to restore the host functional carbonyl group and destroy the virus' amine group it must need for further vegetative integrations. The virus is left harmless then, and there are other indications that it is oxidatively fragmented and completely destroyed as well. It appears too from our aftosa experiments that after recovery following the treatment, the animal is not subject to further infection, as the annually recurring epidemics of aftosa do not affect the cured animals, but do attack the new animals brought into the herd, if they are not treated. Our hypothetical azomethine bonding is again indicated in the cure of Rabies since here too host cell function is returned while the virus is made harmless, as in the cures of Anterior Poliomyelitis with extensive paralysis and atrophy, in cinomose, in the final phases of Swine-Pest, and in Newcastle's disease in chickens, in all of which non-reproducing nerve cells are invaded by the virus. And so function can only return when the virus is eliminated and the host cell groups it combines are restored. (Diagram VII and Chapter VII). Among reproducing cells the lactogenic cells and myocardium of aftosa affected cows are restored to function.

Rabies offers a better demonstration, however, since the

disease is one hundred percent fatal and kills in about four or five days after the onset of symptoms. Its course is definite and cannot be confused with any other disease.

Vaccine Intoxication

One must carefully distinguish between the toxic effects of the vaccine and the inactivation of nerve cells due to actual viral integration with the host cell so as to establish a vegetative parasitism. The intoxication by products of virus disintegration probably has an immunizing effect, and is due to products of virus breakdown that have not been identified as yet. They attack the liver, and in proportion to the damage done there, the recovery will be more or less rapid or transient. Many humans who have received anti-rabic vaccine show these toxic effects. They develop rapidly once they are started and then subside. The nervous system is poisoned. There is dizziness. Incoordination of movements may even occur, and other symtoms of a "Drunk," with nervous excitement. The animal may attack, but humans feel "flighty", etc. There is no true reflex block however, and no real paralysis has taken place. Thus the animal will drink if thirsty. One must remember the old adage that "you may take a horse to water but you cannot always make him drink." The fact the animal will not try to drink means he is not thirsty unless he has had too much pain on former attempts. But under such circumstances if one puts water in his mouth and he cannot swallow, there is true reflex block and nerve cell injury, as occurs in Rabies. The rabid animals, after they develop this inability to swallow due to the nerve cell injury, become dehydrated because they can no longer take in fluids. Dehydrated animals that do not drink and are not able to swallow when water is put in the mouth, can be taken as suffering nerve cell paralysis in such situations as we are discussing here. The intoxicated animal will swallow when the water is put into his mouth as veterinarians know how to do to cause it to be swallowed. The rabid animal will not. The water will run out or into his trachea to set up respiratory spasms.

Once the true nerve cell invasion has taken place the changes are progressive and end up with death in typical fashion. The

toxic effects pass off. Swallowing and chewing centers are first to show reflex block and then the spinal centers. Finally the vital respiratory and heart centers are paralysed and death results. The muscle spasms, torticollis, convulsions and paralyses show up in definite order therefore. According to Rivers and other leading authorities, once true nerve cell invasion by the virus has taken place and real paralytic or reflex block symptoms have developed, the subject is doomed; and never has a case been known to ever have recovered. This is also our experience in the present epidemic, except for the cases we have treated.

Experiment

In April 1955 the Fleury live Rabies virus vaccine, made from the street variety of dog rabies, was used for the protection of a large herd of cattle. Of the 650 animals vaccinated at the Fazenda Indiana near Rio de Janeiro, 600 showed no symptoms of intoxication or rabies. In May however typical rabies developed in 23 animals and they followed the typical course and died in four or five days according to the classical symptomatology and course. We arrived on May 19th and treated such animals as had just come down with the disease, except two, Oceana and Tranca, that were under heavy "Hexa-Meth" treatment. We treated them later when in the final hours of agony. It was too late to help them, and they died. All of the other animals that we treated with 6X dilution carbonyl catalyst showed true dehydration and deglutory reflex paralysis at time of treatment.

There were six far advanced or rather terminal cases in our series, and the rest were well established with deglutory paralysis, an inability to chew, in addition to the toxic excitement. Some showed torticollis, and incoordination of movements, and would fall if pushed and then be unable to get up if not picked up, after which they fell again. This phase was noted after the excitement stage passed and was part of the depressed stage even though spastic paralysis and short convulsions were noted. Any disturbance set off a convulsive attack that accentuated the spasticity. The torticollis persisted throughout the final agony in all untreated animals. But in those we gave the

6X carbonyl treatment, it disappeared before the swallowing reflexes were restored. Here we have another confirmation of actual parasitism, for the sequence of recovery events in our experience in such states is a reversal of the order of the symptoms of the pathogenesis. The first to come are the last to go and the last to come are the first to go. Naturally the longer the pathogenic agent acted, the greater the destruction done and the longer it takes to correct it. There is a disadvantage here since the ability to swallow is so badly needed. Generally death took place in the untreated animals in four or five days after the symptoms started. Our animals treated on the fourth or fifth day were quite dehydrated then, and in the four or five days needed for the restoration of swallowing the dehydration was extreme. This with the starvation of vitamins and trace elements as copper and cobalt so much needed for the recovery process gave the animal a bad deal. Moreover, since animals that had fallen to the ground and showed the deglutory paralysis and torticollis were condemned by the Health Officials for sacrifice and autopsy; they did not receive the nursing care that would have been so welcome. Their normal fate was death.

The rest of the cases were in the second or third day of the infection when treated. The symptoms showed well advanced and progressive integrations of rabies virus with the nervous tissue. Yet they could all walk, but not swallow.

One animal, the calf Iberia, was in the earlier phase of deglutory paralysis when treated, but was sufficiently dehydrated to need subcutaneous infusions of physiological salt solution at the time of treatment and a few days afterward. In this case the deglutory muscles were not all paralysed completely therefore when the injection was given. This case represents a very early phase of viral integration with nerve cells. It is very important since it shows that the drinking power was not fully restored before 84 hours after treatment, since infusions of salt solution were still needed a few days to combat the dehydration. As soon as swallowing showed efficiency such infusions were always stopped. Thus the restoration process takes as long in an early phase of advancement as in the later phases. This will appear in the recoveries in the farthest ad-

vanced cases also. In contrast to this case, one calf showed marked toxic symptom but no deglutory paralysis, and recovered spontaneously as all of the merely toxic cases do. In that case there was no true viral integration with nerve cells therefore. In another neighboring fazenda several highly toxic cases were observed. One such case was excited enough to attack. But no reflex block was observed in any. They recovered spontaneously. The veterinary surgeon of the Fazenda Indiana informed me that no animal that showed deglutory difficulty ever recovered except those that we treated. Thus the status of the cases reported here is established as true rabid infections. The control cases that were autopsied showed Negri bodies, and the macerated brains when injected into mice gave true rabies infections. The sections of their brains showed the Negri bodies also, according to the laboratory reports given us.

Fortunately we were able to treat animals in every stage of advancement of the disease, and have controls in each stage, as well as the 23 animals that took sick and died up to the time of our visit. We arranged several types of collateral controls as well; and in this we were aided by circumstances.

The total recoveries of treated animals with definite nerve cell-virus integrations were eleven out of thirteen that received the one to a million dilution of the carbonyls, and could be counted as legitimate material for the test. This gives a close to 85% cure rate. The names of the cured animals are, Iberia, Paina, Idioma, Maravilha, Apa, Oferta, Trama, Docura, Vila, Jupuboa and Tiara. The two cases that died on the same dosage of the same reagent are Oculista and Tella.

This epidemic also gave us an opportunity to confirm and extend previous work with Rabies. Several decades ago one of our colaborators, Dr. Buchen of a small town in Montana was bitten by his horse while rabid from a bite from a rabid coyote. The horse was treated with an injection of a 12X dilution of the serial system of carbonyl groups. The horse recovered, though treated in the terminal phase. After Dr. Buchen developed well defined paralysis and convulsions he received the same dosage and recovered completely, too. Our next experience was in Rio where a few mad dogs were treated with favorable results.

Many questions arose as to how to classify this highly fatal virus with regard to molecular configuration and for ability to make the parasitic azomethine double bond. Thus we desired to compare killer viruses with regard to tissues specificity, and the type of electronic activation the dehydrogenator carbonyl group must receive to be effective, aside from its oxidation-reduction potential.

We set up one experiment with eight animals. They were divided into two groups.

In the first group, we treated three average advanced cases of well established rabies with 7 cc. of a 6X dilution of diphenoquinone. This was done to confirm the lack of effect of the quinone structure in rabies. Sometime ago, we had found that the quinone had no effect in dogs. These three cases died as if not treated at all, just like other controls of a similar classification.

The second group consisted of five animals divided in two sub groups (a) and (b).

Sub-group (a): A dose limit was tried on two calves down in the terminal stage of rabies. They were gvien 10 cc. of a 15X dilution of the serial system of carbonyl groups. They both died. One died eight minutes after treatment and the other one-half hour later. Thus there was no effect from the high diluion and they can be taken as controls.

Sub-group (b): Two calves of the terminal stage, that appeared about the same as the two that died, were given 10 cc. of a 6X dilution of the serial system of carbonyl groups. They recovered fully, the symptoms leaving in the reverse order to their coming. Swallowing paralysis disappeared last. The last calf in the terminal phase was found on the ground unable to get up or swallow, had torticollis and the spastic effects. It was also blind at the time of treatment, and had been sick for maybe four days. After treatment it slowly improved so it could get up and stagger about and drink well. Thus it was improving with the dislocation of the parasite from the host cells, when it got caught in a barb wire fence and cut its neck very badly. Liberal applications of methylene blue were poured into the wound. Methylene blue happens to be an antidote to our treatment. One day later, the condition retrogressed, and on the tenth day

after treatment the animal died. This case cannot be counted in with the test for efficacy, as the antidote wiped out the progress started by the carbonyl treatment. The free radicals on which the recovery process depends are absorbed by the Methylene Blue. Thus we also have an indication that the recovery process in rabies is of the same character as in other diseases.

Therefore, of the far advanced or terminal cases, five in number, two made full recoveries on the one to a million dilution of the reagent, the serial system of carbonyl groups with free radical terminals. The other three may be counted as controls of different types, two of the ultrahigh dilution type and one of the antidote type. The three that were moderately advanced and received the quinone may be taken as controls too as in this instance that particular type of molecule is without effect. We thus also confirmed our previous experience with quinones in rabies. The serial system of carbonyl groups, 6X dilution, cured two out of three, including the fifth calf which was subsequently treated with Methylene blue; while controls only lived minutes or hours after their placebo treatments, and all five were in the same terminal condition at the time of treatment so far as anyone could tell.

What The Experiment Taught

The recovery course in Rabies comprises two principle periods. One of about seventy two hours in which the patient is apparently stationary, and not passing on further toward death. Most of our animals would have deteriorated rapidly and died before this had they not been treated with an effective reagent. So the stationary phase can be taken as a sign that recovery is going on. This is followed by the second stage of the recovery. The symptoms are aggravated, and one not experienced in the matter would conclude the animals are getting worse. We interpret the stationary phase as that in which the virus is undergoing dehydrogenation, and the short aggravation phase as that wherein the virus is undergoing oxidative fragmentation with energy returning over the azomethine bond or bridge into the reconstructive processes of the host cell—just the reverse of what took place when it passed on to the virus. Reconstruction is thus going on as energy is pouring back into the functional

mechanism so the reflexes concerned are exaggerated and the situation looks worse. One cannot blame the Health officer for wanting the animals to be sacrificed and relieved of their misery. However, this phase is so short that when taking place over night, one simply finds the calf up and about the next day eating and drinking diligently, or slowly recuperating when the nerve cell injury had been very far advanced. After the passage of energy from the combustion of the virus into the host cell functional mechanism is finished, the azomethine bridge is cut leaving a carbonyl group in the host cell functional mechanism with which it conducts its functions as before the infection took place and the virus has lost its amine group as illustrated by Diagram VII, according to our analysis. The disease and its cause are removed therefore. The cure is complete.

This experiment showed also that the quinone type of structure does not serve in Rabies. In the three cases treated with quinone, the dipheno-quinone was used in the one to a million dilution. Perhaps some other dilution may have given results, but we have no proofs as yet. The effect of dilution in creating strain in the more mobile bonds deserves consideration. However, the fact that the carbonyl group of dipheno-quinone with its high oxidation reduction potential is not effective as a dehydrogenator in Rabies begs for explanation. Our simplest explanation, though others may be better, is that the carbonyl reagent must integrate with the host cell energy producing mechanism, as the virus has already done, that all three occupy the same field of action within the mitochondrial particle. Further, the configuration of the virus structure will necessarily change the pattern of the host cell configuration. The change thus set up will facilitate the admission of one reagent or other preferentially, especially in the plane that permits the carbonyl group to attack the virus perpendicularly to the plane of the double bond and the carbon atom conjugated alpha thereto that carries the hydrogen atom that must be removed to start the oxidation chain.

Further, for union with the host cell functional mechanism, the reagent must present a free radical to add to one of its double bonds or free radicals. Long chains of carbonyl groups with

free radical terminals fit the requirements for oxidation of the integrated Rabies virus. The quinone structure serves well in Swine Pest, one of its more polarized double bonds offering the means of ligation with the host cell functional mechanism, as well as the offer of a free radical while expressing its rich resonance. Here the dipheno-quinone is structurally more proficient, and hence here too it serves best therapeutically. One may therefore classify virus diseases with reference to their response to carbonyl groups activated by electrons received from ethylene linkages, or from serial systems of carbonyl groups, and as these are possessed by different molecules that offer host cell ligation facilities in various manners.

The same factors no doubt control the specificities of the same reagents in the various types of cancer. For example, we have found the quinone structures "specifically" curative in primary diffuse adenocarcinoma of the liver, and in cancer of the pancreas, that widely invades the stomach, the liver, and the lymphatic system. On the other hand the quinone structure does not show a good record in skin and brain or visceral cancer in general. The serial systems of carbonyl groups were found more satisfactory here. It must be recalled too that the separated ring structures serve as carcinogens preeminently in liver cancer, as acetyl aminofluorene, and the methyl-amino-azobenzenes. So the liver seems to admit the separated ring structures into integration more readily than other tissues do. This seems true for the curative quinone rings too. The steric facilities need much more investigation in neoplastic and viral diseases, one may see. Nothing has been done on such matters as yet, except for our own efforts.

Complicating Factors

There were a few complicating factors that adversely influenced the recovery percentage and these will have to be considered.

(a) **Nutrition**—Aside from the first animal treated in the earliest stage where swallowing paralysis was only starting, and which was restored to normal in three days, the rest could not swallow or masticate and so they rapidly dehydrated, and the malnutrition with its absence of trace elements as copper co-

balt and molybdenum that are absent from the soil hindered the recovery process. The cold rains with the animals lying on the cold wet ground, one of them in a deep ditch for days nearly drowning and unable to make a controlled movement, added to the hindrance to recovery. No one could suggest that the good nursing cured them, even though the veterinary of the fazenda did all that kindness could accomplish.

(b) **Short needles**—Not knowing that the Zebu has a thick subcutaneous tissue that droops and the skin itself is not too elastic, half the needles we used were No. 16 bore and ¾ or one inch long. The other half were two inches long. The short needles evidently did not deliver the reagent into the muscle in all cases and these when injected with a second dose using the longer needle responded well. We may have lost one of our cases by use of the short needle as one died quickly as if not treated.

(c) **Confusion**—The veterinarian who served as technician in preparing the vaccine was not a specialized virologist, but was no doubt expert in the routine of producing the vaccine. He was also placed in charge of the epidemic as the Health Officer for Rio. This was unfortunate as he took the affair too much to heart as if he were responsible for the deaths of the animals instead of chalking it up to the whims of the virus as any veteran seasoned virologist would do. He understood too that legal procedures would be started to recover damages for the loss, and took to the defense of his vaccine by claiming that it contained no rabies virus, although autopsies of the animals that died, including my seven control cases all showed the Negri bodies, and their brain macerates injected into mice produced rabies in all the mice, and the brain sections of the mice all demonstrated the Negri bodies. This work was done by the pathologists of the Rural University, the government Agriculture College. Our effort to explain the recovery reactions to the health officer fell on deaf ears, and as he had the right to condemn any animals he wished for sacrifice and autopsy, this was a little dangerous as the following experience shows. It may have reduced our cure percentage some.

The incident was the following, and depended on the fact that Rabies is accepted as 100% fatal. So when the animals

reach the terminal agony they are sacrificed to shorten their misery and to make better biopsy facilities available. This was practiced on the first group as they reached the terminal phase after the first number had died in typical rabies fashion and established the course. It was used on some of our earliest cases that did not recover right away. As the terminal cases take longer to recover, those treated when they were found in the terminal phase after being condemned for sacrifice by the Health Officer, naturally must require at least four days to undo the damage done by the virus. This seemed too long for one who did not understand the therapy. So one such calf that had been condemned for slaughter by the Health Officer, and was treated when it was lying on the ground and paralysed, unable to chew or swallow, and unable to make coordinated voluntary movements, with torticollis, and in about the same condition as the two that died within an hour after being treated, was still lying on the ground 84 hours after treatment, and apparently not making any headway so the Health officer had it dragged to the truck to be taken away for sacrifice and autopsy. But as it was in a position to be lifted into the truck, it kicked up a fuss and tried to get away. The veterinarian of the Fazenda happened by, observed good coordination in its movements, and made other tests for recovery. He found it was able to swallow and chew, so he led it back to the pasture and let it live. It quickly ate its way into good health, and is cured. Thereafter the rest of the treated animals were allowed to make recoveries without interference, or as in the case of the wounded cow to die. Autopsies and Inoculation tests proved the disease to be rabies. Negri bodies were demonstrated in the inoculated mice.

Here are two 100% fatal virus diseases, Rabies that is neurotropic, and Swine Pest that is viscerotropic, that kill regularly in 4 or 5 days, which we have found from 60 to 100% curable even in the terminal stages. In this far advanced state, the virus is not only integrated with the host cell, but has built up its pro-progeny at the expense of the host cell, which now is far along in its destructive dissolution, when the oxidation catalyst is administered. Since the host cell is undergoing full reconstruction while the virus is undergoing oxidative dissolu-

tion and perhaps depolymerization concomitantly, the pathological process is fully reversed, and the energy exchanges are specific between the two parties. The reciprocity is specific, with the treatment reagent entering the process as the specific reversing factor. The relationships are therefore most fundamental, and hence must also be simple, as we have presented them. Such virus parasitism is thus curable on a logical basis, when it was never curable before.

The basic chemistry of the vital unit mentioned earlier is supportive. Thus the phenomena of the double bond will depend upon the nature of its substituents. With an amine group as a terminal substituent the electron drift over the ethylene linkage is away from the amine group and toward the other end where we assume the virus resides. Virus support is thus favored. If a carbonyl group is the substituent, on the other hand, the electrons tend to concentrate at the carbonyl group, making of it an active dehydrogenator, or oxidation initiator. The parasite is devoid thereof, while the host cell requires it in its energy production. The condensation of the parasite's amine group with the host cell's carbonyl group to form the azomethine double bond, will permit energy to pass either way with equal facility, depending on the position of the energy source and the position of its utilization. Thus when the virus progeny is being built up at the expense of the host cell the energy passes to the parasite, but when the parasite is undergoing oxidative destruction, energy returns to the host cell where it provides for the latter's reconstruction. The azomethine bridge is thus neutral and serves not only the energy transfers, but also the disligation of the virus with destruction of its amine group, while the host cell carbonyl group is being restored. Provisions for rapid recovery thus lay in the basic chemistry of the vital unit as we picture it.

AFTOSA

(Hoof and Mouth Disease)

This disease occurs in such great variety of virulence that only by treating many herds under different circumstances can efficiency be tested. But a carefully controlled experiment as

summarized below, using a known virus, bred to the peak of deadliness of the cardiotropic variety will show how the virus can be separated from the cell even weeks after it has produced a deadly myocarditis.

This same deadly type of virus, occurring naturally, was encountered by us several times in epimedics that threatened to wipe out herds of very costly cattle. On October 14, 1949, at the Instituto Quinze de Novembro, a veterinarian college with 59 head of cattle and 200 pigs, the professor in charge was afraid that his herd would be wiped out by this virus. When we arrived, five animals had already died. This was a few hours after the epidemic had started. Others were lying on the ground, unable to get up and soon would be dead. We treated the 54 animals that were still alive. Of these, 7 were newborn, 15 were calves, 17 were young bulls and 15 were cows. Two-thirds were very favorable hosts for the most destructive action of the virus.

Results—two cows and one calf died of the disease. All others recovered and no new infections developed.

Of 200 pigs, 35 were adults, and 165 were young. 167 were well established cases of aftosa, while 33 were symptom-free. All were treated.

Results—4 pigs died of the infection giving a cure percentage of 98% and as no new cases developed a prevention percentage of 100%.

The following year aftosa struck the institute again and ruined the new cows only, that were brought in the herd. The animals we had treated the year before did not develop the disease except a few cases that took it mildly and soon cleared up. So the protection was practically 100% and has so remained for the several years that followed.

In another observation with a milder infection that always killed some members of the herd, but not over ten percent, was at the Rural University, the government Agriculture school, in June 1951, and reported in Veterinaria, no. 1, p. 75, 1951, by Dr. Adalberto da Silva Carneiro, the virus type was identified as "C" with 68 cows in the herd and several not showing symptoms. All recovered as inspection showed four days later and

the lactation which was suppressed had returned to full amount within 24 hours, following one dose of the remedy. But one calf required two doses. No new infections developed and none died. This is the usual type of virus encountered and the results are the same in all other experiments.

The controlled experiment was done on thirty young bull calves, all the same weight and age. Ten were inoculated with the virus and held for controls; ten were given preliminary "protective" vaccine treatment before the virus was inoculated, and ten were treated with the carbonyl compounds three days after the virus inoculation. The virus used was one that regularly killed 80% of animals by heart damage in from 4 to 9 days. The rest that did not die right away, did so sometime later of chronic myocarditis. The experts at the Institution who prepared the virus and the vaccine they made from it, knew their virus to be a standard product and what it universally did in the dose used. We treated the first ten with a dilution of one to a trillion to establish a dosage of the highest limit of dilution; and had one death the fourth day with nine cures. The vaccine protected animals showed four deaths the same day as one treated calf died. The rest were exceedingly sick with myocarditis and the controls were judged to die quickly at this time, and as there were no facilities for cremating so many animals at one time we were asked by the director to treat the controls as they would die otherwise. We gave a dilution of one to a million this time and the curative response in all was immediate and complete so that here, although the myocarditis was advanced to a near fatality point the disligation of the virus was fully established and the heart muscle restored to normal action in a matter of hours or days. Later one vaccinated animal that survived was very ill with myocarditis and had an enormous aftosa abscess that covered his right side. Such animals always die, but we treated this one and obtained a cure. The results,—90% on the 12x dilution, and 100% on the 6x dilution were cured using the serial systems of carbonyl groups.

Many small and large herds were treated with animals that could no longer rise from the ground all of which recovered, if the animals were left in the field. **However, where ammonia**

saturated the air that was breathed in the barn yard or stable the results were disappointing. The same holds true for any infection including brucellosis in the animals we have observed. **Cresols also interfere.**

CINOMOSE

The first official test done in the army Hospital for Small Animals, at Rio de Janeiro yielded a cure percentage of the animals seen through to the end of the experiment, of 80% including the first 17 which died while trying to arrive at a proper dosage, (Carneiro, and da Souza, Veterinaria, Ano IV, Num. I, p. 21, 1950). Thus with these trial animals left out, the cure percentage was 95% at least when the correct dosage was used. And this has been substantiated by further observations by a number of veterinarians in private practice. It includes the serious nervous system infections which otherwise are always fatal. Closely observed cases of this class show a cure percentage of 80 in private practice even when paralysis was established for weeks and was widespread. Altogether several hundred dogs have been treated. The separation of the integrated virus is thus established in other deadly virus infections besides Infantile Paralysis. Such a feat has never been accomplished to date by any other therapy. While the serial systems of carbonyl groups as used in the trials just described have yielded the best results in Cinomose, diphenoquinone has done well consistently here, but the recovery course is much slower and does not complete itself at times unless a dose of the former reagent is used. This is especially true in the paralysed cases. But in Swine Pest it has served well right along, and benzoquinone has served well here too. This would indicate that in Swine Pest there is much more toxin circulating not combined with the tissue cells than in cinomose, and hence the facilities for adding toxic free radicals offered by the many double bonds gives the advantage. Our first discouragement with diphenoquinone came with repeating the dose where a block to the recovery was had instead of the boost that was desired. We learned thereby that the second dose must be very diluted if given at all.

The separation of the virus from nerve cells, and its

separation from heart muscle cells to which it had acquired a fatal affinity in aftosa, places the value of the oxidation mechanism in curative as well as protective therapy as very high. We can not escape the fact that living processes in all aerobic life depend upon the chemistry of oxygen in its molecular form. The secretion of milk too, by animals as they recover from aftosa after this treatment is completely back to normal and takes place immediately, that is within twenty-four hours, or sooner. This is established in many herds about Rio. (Carneiro da Silva, Veterinaria, Ano V, No. I, p. 76, 1951). The return of function is thus prompt with separation of the virus whereas without the carbonyl catalyst treatment it scarcely ever returns and if it does, it is only slight. The manner of union of virus and host cell substance in any instance then seems to be in conformity with that shown in our diagrams, and takes place with the energy producing mechanism. Since the virus on separation is no longer infectious, its chemistry may well be changed as we suggested by acquisition of a carbonyl group which indeed may serve it in initiating oxidation chains for its own energy production and function so that it need not and can not again become parasitic. The same conclusions seem justified from the results of the cure of mastitis in animals where the most virulent streptococci and staphylococci may increase in number during the healing of gangrenous areas, and while the toxic state of the animal is rapidly disappearing. Here the increase appears to follow the amount of tissue debris that must be removed.

The recovery percentages in animals suffers the same handicaps as in humans except that animals are under more thorough dietary control and can be held on a vegetarian diet. We find some dogs after taking vegetables only and some milk for a few weeks, refuse to eat meat any more, and they get along very well indeed. Habitual use of narcotics and depressants, or "calming" drugs are eliminated and so when the veterinary has not overdone with sulpha drugs, ACTH and other modern remedies, the recovery rates should be and are better than in humans where they will cheat on the diet, or on smoking, and call for aspirin and barbiturates in all the different

forms that are offered. Besides, the animal has not been ruining his power to carry oxygen by the blood, or the ability of the tissues to use oxygen, by the use of such medications.

SWINE PEST

In the treatment of Swine Pest (Hog Cholera) the same difficulties were met. Cresol antiseptics and other affairs have interfered as much as 100%, as in two experiments in the pens of the Ministry of Agriculture Maracana station. But in the Ministry's outer stations as at Deodora, and in private farms where such interferences were avoided and only lime was used to clean the pens, the results have run from 60% to 100% cures repeatedly even in the most severe epidemics. Usually one dose proved sufficient but a second dose hastened recovery so that it took three to five days to obtain true and lasting cures. Many of the cured animals could not rise from the ground or eat or drink when treated. That is, they were in the terminal stage when they received the first dose. A second dose given 84 hours later met them on their feet able to take nutrition, but still showing some cramps and traces of hemorrhages into the abdomenal skin. The second dose boosted the recovery rate very remarkably, and should always be employed in cases of this type. The second dose (12x) is always more dilute than the first, (6x) on the decimal homeopathic scale. Most of the herds were small. Since the virus is exceedingly important and *vicious,* a complete discussion is planned for the complete text.

Our observation in animal diseases convince us also that all pathology follows the same pattern. (See Chapter VII). In Swine Pest all tissues of the body are attacked with rapid fatality, an apparently lytic type of virus-host cell integration, but only the pig is attacked. In cinomose the virus may be specific for either the respiratory, the intestinal or the nervous systems, or for all three, and only a few species are attacked, as the cat and dog. In Poliomyelitis, many species are attacked, but only certain cells of the nervous system. In both cinomose and Poliomyelitis both the lytic and symbiotic types of integration are usual, and this is true for aftosa which while usually

symbiotic, may be violently lytic to the myocardium before hoof and mouth symptoms can develop.

Since in these and all other virus infections we have encountered, the survival factor can be replaced by a synthetic atomic arrangement, the carbonyl group, one feels justified in concluding that the survival factor is the same mechanism for all tissues. Specificity for species or tissue is an entirely different matter. The antagonists to the operation of the survival factor have no influence on specificity in any, while they block survival factor activity in all. They are carbonyl inactivators and double bond additives. Specificity of the virus depends upon its protein structure, while specificity of the reagent depend upon steric advantages or hindrances with reference to its union with the host functional mechanism in the plane that permits dehydrogenation of the virus.

Thus in swine pest the serial system of carbonyl groups proved inactive entirely, and the quinone type from 90% to 100% active (diphenoquinone), or 60% active (benzoquinone) in various epidemics. Though the disease is always fatal in 4 or 5 days, in no two epidemics were conditions exactly alike. Tabulation of the various experiments would therefore not mean much. The time of instituting the treatment with regard to the progress of the epidemic, whether early or late, is of no importance. Thus it might be argued that though the infection is one hundred percent fatal in 4 or 5 days, those that died first were less resistant, and those showing symptoms last are the most resistent and would give the 100% cure rate more consistently. This is not our experience, for we have treated the pig that survived in a herd the day after seventeen had just died. Though this pig was the last to come down with the infection, the disease developed at the same rapid rate as in the others, and one injection brought full recovery in the same time as other pigs treated in neighboring farms the same day, when the epidemics were just starting in these farms. One such farm had four pigs in about the third day of infection, and were all equally advanced. One was a very small pig (a runt) that was nearly dead, it had not eaten or taken water all this time and died in a few hours after receiving the injection. The

others all recovered. Another neighboring farm had four with the infection, three in the second day perhaps. We treated the three that appeard worse, and left the other for the control. On returning three days later the treated pigs were about cured and eating well and without fever. The control pig was quite advanced and would probably live another day. We treated it and two other neighbors' pigs that were the first to come down with the disease in their herds. All recovered in due time.

In this group there were nine pigs including the pig that was treated after its seventeen mates had died and it was far advanced in the disease. Of these eight recovered, giving an 88% cure rate. This happened at Santa Cruz in the first week of July 1956. A year earlier we treated five pigs with advanced Swine-Pest in Rio. They were part of a herd of ten, five of which died the day before. The diagnosis here as in the other epidemic mentioned was made by the characteristic symptoms, clinical courses and autopsy findings. Only one of these five could walk. The others were down in the last stage. All were given the dipheno-quinone. Two injections were given; the first injection was a 6x dilution, and the second injection which was given 84 hours later was a 12x dilution of dipheno-quinone. All five of the pigs recovered, thus giving us a recovery rate of 100% in this group.

The age of the animal makes no difference, but cleanliness does. The diagnoses were made by the veterinary surgeons of the Department of Agriculture or of the city veterinary services where the animals were treated. No case was treated without all the cardinal signs and symptoms being present, such as the scleral and cutaneous hemorrhages, the pneumonia, the intestinal changes, the very high fever, or the specific test, and autopsy findings of deaths close by and at the same time. We always arrived late enough for all the data required for a firm diagnosis, after the animals had refused food or drink for several days. The recovery course comprised a period of 72 hours where improvement is only slight, but drink and food is again taken. The pneumonia is still present and the cutaneous hemorrhages are improved some. Then twelve hours of aggravation, is followed by rapid improvement. This is the same course seen in

Rabies recoveries, and its interpretation is given in that section. The cure takes about five days.

THE FATE OF THE VIRUS

The fate of the virus has not been fully established. Clinical evidence has indicated three possibilities. The first possibility is that a part of the virus ligated with the host cell may be burned off leaving a considerable portion still occupying the functional carbonyl group. To compensate for this the host cell constructs another functional carbonyl group. This is illustrated by the type III integration we have suggested, which so changes the steric setup of the host cell that another virus can not ligate with it. A second possibility is the complete oxidation of the virus with restoration of the host cell. This would be an involution process that returned the energy, taken from the host cell during the virus vegetation, back to the host cell and would be used in the latters reconstruction. A restoration of the host functional carbonyl group would be accomplished and the virus or rather provirus would be destroyed. The host immunity would possibly envolve a change in steric setup as the reconstruction came about in a different direction than the usual. The third possibility is the oxidation of the virus off at or adjacent to the azomethine bond. As the virus is separated from the host cell or burned off all at once, the virus would be left with a carbonyl terminal instead of its amine group, and thus it could make no more ligations with the host cell. It is possible that the virus could now conduct its own oxidations for its survival. The host cell is left with its functional carbonyl group restored and is no worse for the experience. Immunity is gained probably through steric change, though other probabilities exist for this also. After treatment oxygen use is increased and area production is diminished in neoplasia. This confirms the azomethine ligation with the pathogen.

CHAPTER XII

INCOMPLETE COMBUSTION BURNS

Products of incomplete combustion caused by suddenly elevated temperatures, from steam, or fire or hot metals demonstrate much the same tissue changes as do the incompletely combusted products of tissue and of germ metabolisms. They offer the toxicities that prove fatal, through gelling of the blood and tissue colloids as well as by direct embolism. This gelling of the blood that prevents its flow through the smallest vessels to cause infarctions, resembles also the situation in coronary occlusion and in cerebral apoplexy. When the oxidation catalysts are used before the vascular wall is completely starved of oxygen and fragments to permit hemorrhage, the dispersion of the colloids is restored and the blood flows on to supply the oxygen and other things the tissues need so that apoplexy and occlusions are prevented. And even after such changes have taken place the treatment has restored the non-toxic normally dispersed state of the colloids so that lesions that exist do not get worse and besides Nature has every opportunity to correct the damage done. Some severe cases of established apoplexy have been restored to a close normal in this way instead of suffering profound invalidism moving on toward death through repetition of the pathology. We submit two cases of burns to illustrate that the incompletely combusted products can be burned out of the way so the usual destructive changes are avoided.

C. B., Nov. 7, 1929, while repairing an automobile was severely burned by a gasoline flame that caused extreme erythema, superficial desquamation and terrific pain, but no immediate visible charring of the tissues. An injection of the serial system of carbonyl groups with application of a high dilution over the burned surface brought immediate relief and rapid healing without tissue destruction or scar.

Mrs. L. T., Sept. 16, 1948, opened a pressure cooker under pressure. The hot soup struck her abdomen covered by a thin cotton cloth only, and caused severe pain and shock. Benzo-