

6 Evaluation of Exposure and Hazard in Chemical Catastrophes

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6.1 INTRODUCTION

Every contemporary, technically developed society is characterized by a high degree of chemical utilization. The diversity and quantity of chemicals routinely used in industry, agriculture and everyday life has enormously increased. At the same time, the biological effect of these compounds, especially their long-term action, is only partly known.

Two new global risks from chemical compounds have appeared in connection with their large-scale production. The first is the ecotoxicologic hazard stemming from the permanent and ubiquitous penetration of very small concentrations of toxins into the bodies of a great number of persons. The product of low probability of impairment multiplied by millions of exposed individuals produces, however, numbers of simultaneously impaired individuals corresponding to the notion of a diffuse and chronic catastrophe. The second risk is associated with the acute escape of large volumes of toxic substances into the environment at chemical accidents. The probability of an accident is small but, should it arise, a great intensity of toxic effects in an extensive area of the cloud of highly concentrated chemical suddenly develops. The product of the great probability of effect and the large number of affected individuals in densely populated areas creates the danger of a large number of victims. The escape of toxic chemical from containers which in small-scale production would be negligible can, at the current capacity of production, lead to acute chemical disasters endangering life over a radius of tens of kilometres from the epicentre.

The most simple strategy for human protection from chemical danger is not to produce and not to use highly toxic and biologically persistent substances. Plants should be placed to keep large agglomerations of people outside the radius of impact of accidents or to size the production in such a way that even the maximum escape could not injure the residential settlements. Although there are strict rules about maximally tolerated amounts of dangerous substances

in chemical laboratories with perfect control of operations, containers with thousands of tonnes of toxic chemicals persist in industrial plants.

The necessary basic methods are already available for reducing the ecotoxicologic hazard and for modelling the effects of substances chronically penetrating into the environment. However, these methods can be applied only to a limited extent for predicting acute chemical accidents (Berthouex and Rudd, 1977; Wark and Warner, 1976). It appears that in this area, too, a systemic approach will be basic (Emery, 1972; Shaefer, 1974; Jeffers, 1978; Gigoh van, 1978). The new quality of acute toxic risk will require a complex and interdisciplinary approach to all aspects involved, including the psychologic and sociologic ones. Important also will be the theory of reliability as well as the ageing of equipment which are connected with the occurrence of accidents (Gerzbach and Kordonskij, 1966; Kapur and Lamberson, 1977; Collins, 1981).

The probability of chemical accidents at the present level of technical and control mechanisms is very low but never zero. Recent disasters in Seveso and Bhopal proved that point and aroused interest in the problem. The limited reliability of technical equipment is borne out also by accidents in space research where the level of reliability of machinery reaches the peak of human capability.

6.2 CHEMICAL DISASTER

Large-scale technologic disasters are a phenomenon of our times. Their prerequisite is the accumulation of destructive energy and of a great number of people in the same place (Roding *et al.*, 1985). With regard to medical care, a new discipline, disaster medicine, has been established oriented to professionally efficient handling of human casualties in disaster situations (Peter *et al.*, 1984; Lanz and Rossetti, 1980).

The term 'disaster' is usually understood as a great calamity at which many persons perish; according to international classification, at least 50. An event in which only one or a few persons are the victims is assessed as an accident. The criterion of the number of dead permits a classification of the event, but it impairs the use of the results obtained for assessing rescue measures in practice. It is analogous to the utilization of the post-mortem findings for the treatment of the dead. A retrospective criterion limits the quantification of decision making and optimization of rescue operations.

For these reasons, it would be more appropriate to regard as a basic criterion of disaster the number of endangered persons. Characteristic of a catastrophic situation is that the need for aid surpasses the normal capacity to deliver medical and technical assistance. People involved on a mass scale have a lesser chance of receiving optimal or standard care than in cases where only few are affected in the same way.

Estimates of the number of endangered individuals may serve as the basis for

decisions whether to employ special means for evacuation, rescue, and therapeutic requirements. Instead of the number of 50 persons, it would appear more suitable to declare a catastrophic situation already at a threat to ten persons. This is the number of individuals with which the crew of an Emergency Medical Service (EMS) ambulance can effectively cope. This number corresponds to the limit of injured at the collision of two cars (Spacek, 1981).

A chemical disaster is a situation where the presence of toxic chemicals in the environment endangers or damages, acutely or through late effects, the life or health of a greater number of persons. The key characteristics of chemical disasters differ from catastrophes due to physical causes (White, 1974). They are more diffuse in space and time. They are closer to radiation disasters, persistent substances, or epidemics. The toxic effect in comparison to physical damage has a much greater variety of symptoms and may be considerably delayed in time.

A chemical disaster is a set of accidents varying in effect on individuals. The summary risk is not merely an additive sum-up of individual risks, but greater. The worst prognosis is due to the mentioned saturation in the capacity of the health service with respect to diagnosis, therapy and evacuation. Organizational and psychologic factors also play a part in rendering first aid among rescuers and the rescued.

The basic axiom from which one has to work in an analysis of the course and efficiency of aid at a chemical disaster is the scientific exploitation of all knowledge and facts derived from its actual course. The objective should be not solely to rescue the endangered or affected persons, but also to view the event as a unique passive experiment carried out by chance. The damage can be partly compensated by retrieving all the experience for more effective prevention and a more rational solution of possible similar situations in the future.

Quantitative information about the effect of toxic agents on the human organism is still insufficient. Worldwide long-term collection of data about the course of accidental intoxication in man is the only way for obtaining toxicometric parameters and for effective treatment. Generalized laws of the spread of toxic substances in individual cases may serve as the basis for construction and correction of models for estimating the ground-level expansion of the substance.

6.3 EXPOSURE, BIOLOGIC EFFECT AND HAZARD

The medical basis for an operative prediction of the severity of the chemical disaster is assessment of the intensity and extensiveness of risk to health in the affected area. This risk is understood as the probable harm to vital functions or danger to the life of individuals due to the action of factors. Risk is a specific case of an adverse biologic effect. The connection between exposure, biologic effect and risk is of a stochastic nature.

The biologic effect of the toxic substance is limited by death of the organism. Between the dose of the absorbed chemical and the probability of a lethal or other selected effect there is a non-linear dependence. The probability course of the dose-related reaction corresponds to a sigmoid curve. For proper transformation the probability of the biologic response can be calculated by linear regression. Currently the basis for predicting an effect is the probit model which has a clearly elaborated statistical methodology (Finney, 1971).

Prediction of the biologic response on the basis of this model (linear two-parametric regression between the logarithm of the dose and the probit value for the given probability) applies, however, only to a limited dose range of approximately ED_{05} to ED_{95} . At lower doses, estimates of effect are repeatedly under-rated whereas at higher ones the effect is over-rated. This limits substantially the extrapolation power of the model for very low doses and concentrations of toxin whose prediction of effect forms the basis for assessing the environmental risk. A better estimate is given by the logit model (Berkson, 1953), but here, too, prediction is not satisfactory. We believe that the solution of the biologically important dose-effect law has been found by us in the binit model (Dolezal, 1985) which provides satisfactory estimates of effects even in areas of very low doses.

6.3.1 Binit Models

The binit model is formulated by a simple regression equation without logarithms of doses, formed by the expression

$$\sqrt{\frac{P_i}{1 - P_i}} - \sqrt{\frac{1 - P_i}{P_i}} = k_1 \sqrt{D_i} - k_2 \sqrt{\frac{1}{D_i}}$$

where P_i is the probability of reaction at a dose D_i and k_1 and k_2 are coefficients.

$$\text{The value } ED_{50} = \frac{-k_2}{k_1} \quad \text{and} \quad K = \sqrt{-k_1 \times k_2}, \text{ where } K \text{ is}$$

the line slope of the structural form of dependence with doses proportionalized with values ED_{50} . Coefficients are calculated by weighted regression. Estimates are calculated on the basis of solving simple quadratic equations. The basic procedure for calculation is described in the cited study. We assume that in the calculation of the risk estimate at low concentrations the binit law will find its justified application.

6.3.2 Risk Assessment

All models of the biologic effect are two-parametric. It is a great pity that the world literature gives for a large number of experimentally tested toxic substances solely the value of one parameter – the median effective or lethal dose, ED_{50} or LD_{50} . However, the steepness of rise of the toxic effect is as important a parameter as the median dose. It would be sufficient to document at least one further effective dose, e.g. ED_{16} on the basis of which it would be possible to calculate the parameter of steepness and thus obtain estimates for the whole range of doses. This applies not only for the one applied parameter but also for other presumed dose–effect laws. Due to insufficient documentation of two-parametric dependence by only one parameter, the essential information yielded by costly biologic experiments and observations gets lost. At the same time the toxic effect of the substance is paradoxically expressed in micromoles of the substance per weight of the organism which with the emergence of computing complications provides no advantage whatsoever.

Besides assessment of reaction in the whole dose range, for estimating the risk of persistent substances, kinetic parameters of the disappearance of substances from the environment must also be known. For these purposes there exists already a well-elaborated mathematical system of pharmaco- and toxicokinetics (Rossum van, 1977; O'Flaherty, 1981). Simple first-order reaction is, however, an exception particularly for lipophilic compounds. The principle of superposition of exponentials is of advantage and can be simply described also graphically (Moghissi *et al.*, 1980). For a long-term estimate of the risk the criterion of the 'last' least steep exponential is suitable.

Substances enter the organism at an exposure which lasts for a certain time. The product of the concentration in air and the time of action of the substance provides the first estimate of the dose that maximally could have entered the organism. At calculations of the rate and volume of respiration the inhaled amount is by about one-third smaller. At a certain simplification, ignoring the mechanism of detoxification, elimination and adaption, the level of exposure could be proportionate to the calculated dose of toxin.

This procedure corresponds to expressing the risk by means of the so-called exponential product expressed as ' $C.t.$ ' according to which at the same product of concentration (C) in air and time of exposure (t) the resultant effect is identical. The dimension of this indicator is $\text{mg min}^{-1} \text{m}^{-3}$. For toxicologically important gases the values of this coefficient are known for a number of effects, including the lethal effect. Instead of the usual expression of effective doses by the symbol ED or LD the symbol $EC.t$ or $LC.t$ is used with the addition of the per cent of the anticipated effect.

Even though there are deviations from this rule, no better procedure is known as yet. On the basis of experimental data or analysis of real situations it is possible to derive graphically or numerically the correlation between the exposure product and effect in a satisfactory range of values. On the principle

of the exposure product one can construct a rather general model of the spatial expansion of the toxic substance.

From the above it follows that the biological effect is the probability function of the dose, the size of the dose penetrating into the organism, and the function of the exposure product. The probability of risk is in the first case identical with the occurrence of the adverse biological effect which represents the risk with a view to the size of exposure. Probability, however, is modified by other factors such as by a preventive reduction of exposure and efficiency of therapy. These influences can be quantified or at least relatively compared.

If we assume that at exposure to a toxic agent of magnitude X the probability of a lethal effect will have the value P_x and standard therapeutic efficiency, including optimal speed of diagnosis, therapy and sufficient means is expressed by the probability of recovery P_T , the value of the summary risk $R = P_x \times (1 - P_y)$.

In the application of preventive measures, including evacuation, we calculate with a lower value of exposure corresponding to the decreased time of the compound's action or its absorption before entering the organism.

Exposure of a great number of individuals will be characterized by the sum of individual risks. In relation to the total number of persons the number of dead or injured can be calculated by estimation. In this case, however, due to the mentioned overtaxing of medical capacity, the value P_T will be reduced. In the case of therapeutic failure the summary risk equals the risk of untreated intoxications.

The logic of assessing risk at acute disasters differs from the evaluation of chronic exposure in the workplace or ecosystem. Taking into account the multiples of excess of maximum permissible concentrations provides no usable information for estimation of consequences. Again it is a matter of simplifying correlation by one parameter.

The MAC, maximal accepted concentration, value represents the limit of an agreed upon rather than a quantified hazard. To express the degree of the increase in risk by means of multiples of MAC excess is invalid and rather obscures assessment of the actual threat. In view of the variability of steepness between the increase of risk and the increase in concentrations the estimate can be very inaccurate. Differences against reality may be of orders of magnitude.

These uncertainties arise also with a standard assessment of the living and working environment on the basis of MAC relations, especially concerning the combined action of toxins. For the reasons indicated we have proposed for these cases, too, direct calculation of the hazard on the basis of regression in relation to exposure at the general criterion of accepted borderline risk. This procedure provides clear-cut estimates of the combined action of the toxic substances. The danger induced by individual factors can be compared and summed (Dolezal, 1984).

Practical calculation of the risk estimate is based on determining the product of concentration and exposure time. According to the previously determined regression correlation between the exposure product and key effect, death, the probability of its occurrence is estimated. This probability is multiplied by the probability of therapeutic failure with treatment of the given intoxication. The resultant value indicates the individual hazard.

For group hazard, an estimate of the mean exposure product in the area where people are present, is calculated. In the same way the probability of effect is calculated, corrected by therapeutic ineffectiveness. By multiplying the number of persons in the group with this value we receive an estimate of the number of affected persons. In case of multiplication with an uncorrected variable, an estimate of the number of affected persons receiving no aid is obtained. Comparison of these values with the *a posteriori* reality permits calculation of the therapeutic efficiency at the event.

6.4 DETERMINATION OF EXPOSURE TO TOXIC AGENTS

The basis for a concrete estimate of the risk in the contaminated area is as accurate as possible determination of exposure. Compared to the routine procedure of occupational health the situation is much more complex. Exposure of a large number of persons must be estimated simultaneously in a limited time. Exposure values may attain high levels and form a basis for crucial therapeutic and organizational decisions. A specific case is the need to define the area with a high threat to life which must be evacuated.

Direct Determination of Concentrations in the Environment on the Basis of Local Monitoring

Analytical methods are well elaborated, sensitive and valid for the majority of toxic substances. Primarily, the air must be analysed by simple and more demanding routine methods (Leithe, 1974). The methods are included in the routine programme of hygienic and plant laboratories. For a rapid decision of evacuation at chemical disasters these methods are, however, slow and provided there is no network monitoring, also not representative. Measurements in the contaminated terrain are difficult and of limited capacity. The data obtained are, however, valuable for controlling exposure estimates obtained by other procedures.

Direct determination of toxic concentrations in the environment is of great value for determining residual concentrations of persistent substances and verification of the hypothesis about spatial movement of the chemical at a disaster site. At chemical accidents there are usually no problems with the

identification of the chemical since this is usually known. The concentration of supratoxic side products can be determined on the basis of precursor concentration, e.g. trichlorophenol for TCDD.

Exposure Tests

The aim of exposure tests is to determine the contact or level of the individual's exposure to the chemical on the basis of its direct determination, definition of the metabolite, or of biochemical changes induced in the organism. These determinations are primarily made in urine, plasma, expired air or saliva. Quantitative determination of low concentrations in biological material usually poses no problems (Teisinger, 1980). Instrumentally demanding methods, like GC, HPLC, etc., are currently routine procedures even in small laboratories. A complication is the frequent tediousness of the methods which restrict the simultaneous analysis of large numbers of samples in the required time limit. From this aspect, simple, sensitive and specific immunochemical analytical methods are promising.

The basic restriction in the predictive validity of these methods for exposure estimates are biological factors. The great variability in results is due to the time kinetics of the substance in the organism and the great inter-individual differences in metabolism, distribution and elimination of the substance. This great variance is even more pronounced in biochemical and enzymologic methods. Determination of reliable control and normal values gives rise to further problems.

Exposure tests do not, as a rule, provide spatially and temporally representative data in the acute phase of a calamity. To withdraw samples from persons with clinically manifest intoxications is of no therapeutic significance and may be ethically contraindicated.

Exposure tests are more meaningful for assessing the individual risk in the period following the accident and for long-term monitoring of the state of the exposed person, and for prevention of late effects of intoxication. They will be of benefit in the monitoring of subclinical impairment in susceptible individuals in marginal zones of the accident. They are also useful in the biomonitoring of exposure in animals caught in the area of the accident.

The extent of usability of exposure tests for estimating exposure at chemical disasters is unsatisfactory. Values obtained should be regarded as semiquantitative. It is well to remember that the discrimination efficiency of the most simple and cheapest procedures is often higher than of complex analytical methods.

The inadequacy of these procedures is illustrated, for example at exposure to a reactive substance like TCDD. A manifest symptom of exposure would be, according to all experimental expectations, impairment of liver tissue. The

lymphoid tissue is also sensitive and TCDD has shown in animal experiments an immunosuppressive effect.

An enhanced excretion of uroporphyrin in persons suspected of exposure to TCDD in the manufacture of herbicides was described first by Bleiberg *et al* (1964). In a long-time study of workers, exposed at a similar production, only in 22% a permanent and in 20% an intermittent increase of porphyrin in urine was found (Jirasek and Kalensky, 1973). Such frequencies are insufficient even for a qualitative assessment of exposure.

Application of a battery of biochemical tests for studying the long-term effects of TCDD exposure following the accident in Seveso again demonstrated very uncertain results. Values fluctuated considerably in both exposed as well as control groups (Mocarelli *et al.*, 1980). Changes were not striking in values of serum alanine-amino-transferase and gamma-glutamy-transferase. Induction of liver microsomal enzymes was confirmed by excretion of glucaric acid in urine (Ideo, 1979). Even though some differences were statistically significant, correlation with exposure was very slight and permitted neither quantification of exposure nor discrimination of endangered persons on a usable level. Abnormal results in liver tests were again found only in about 20% of exposed individuals. This was confirmed also by other authors. It is a paradox that the diagnostic power of the most superior biochemical tests did not surpass discrimination of estimated liver lesions achieved merely with palpation by hand.

The situation was similar at the use of immunologic tests (Sirchia *et al.*, 1980). Ten of these were used in the analysis of children in Seveso and only three showed significant differences in comparison to controls, i.e. the complementary haemolytic activity test, the PHS test, lymphocytary response to phytohaemagglutinin, and routine lymphocyte count in peripheral blood. Surprising, however, was the finding of a higher immune response instead of the anticipated immunosuppression.

Apparently it will not be easy to find usable exposure tests. Chemi-(CL) and bioluminescent (BL) methods could be very useful for their high sensitivity and simplicity. Determination of free radicals and/or their products could be performed by means of CL and BL and seems to be prospective (Boeckx, 1984). Detection of activity of detoxifying enzymes, SOD, GSH, peroxidases, etc., could be very promising.

The simplest and cheapest procedures, like monitoring the blood picture and basic clinical examinations, may be more helpful. Likewise determination of haemolysis, tests of methaemoglobin and other haemoglobin derivatives at inhalation intoxications may be used.

Nowadays, with the omnipresence of organophosphates, simple pH-metric determination of cholinesterase becomes increasingly significant.

Evaluation of Exposure to Toxicants on the Basis of Clinical Observation and Examination is a Classical Semiquantitative Procedure

The nature of symptoms, their intensity and frequency correspond to the severity of intoxication and thus to the level of exposure. The validity and speed of diagnosis by an experienced physician competes effectively with results yielded by demanding laboratory methods.

The basis for the required quantified assessment of exposure on this principle could be the establishment of standard examination procedures with a high degree of use of algorithms and formulation of suitable scales for expressing the frequency and intensity of symptoms. With long-term collection and generalization of data about intoxications on an international level, it would be possible to create the basis for a quantitative prognosis of intoxication and thus also an estimate of exposure.

But this, too, gives rise to a number of problems. Even the frequency of occurrence of chloracne, which indicates the degree of TCDD intoxication, shows in spite of its specificity small discriminating power. Between the second and sixth week following the accident in Seveso the most affected zone A showed a frequency of chloracne in 14.5% of children and 0.63% of adults. In other zones B and R there were no such findings. Six months later further cases of late chloracne occurred which, however, did not correlate with the anticipated exposure according to TCDD concentrations in the environment. From the analysis, it follows that determination of the degree of exposure on the basis of the appearance of acne is not very reliable and moreover that the absence of acne does not exclude exposure.

For an indication of exposure to a number of industrial chemicals, neurologic symptoms are typical, a fact which was repeatedly confirmed for TCDD. As a rule banal neurasthenic difficulties arise – anorexia, weakness, unspecific pain, etc. It would be of great benefit to document these symptoms by means of a suitable international questionnaire even to the exclusion of exposure.

In clinical examinations of intoxications, too, one should expect a great variability of results which, moreover, are merely of a semiquantitative nature. None the less, there is nothing to prevent the test, at least at sub-acute or long-term monitoring of exposed persons, of all the promising methods devised by classical and modern medicine. Even if their use is dubious for exposure estimates they may be of value for the control and assessment of therapeutic efficiency. The interdisciplinary approach may give valid estimates where methodologically isolated approaches fail in prediction.

The basis problem is, however, to estimate exposure in a large number of intoxicated individuals directly in the contaminated area. In the acute phase of the disaster it must be assumed that it will be possible to classify the state of those affected and in danger with the most primitive methods only. The physician will have to decide about some vital therapeutic intervention or preference of evacuation after a few seconds of observation.

For critical situations in the field it would be useful to develop at least a fragmentary system of telemetric documentation of the situation of the decisions, and of the state of persons, which would permit the permanent consultation and correction of treatment from the medical centre and possibly also retrospective verification of primary estimates of exposure from the field. A similar system was tested at the work of an EMS squad without a doctor (Hirschmann, 1974).

Factors of medical surprise will play a role in every chemical disaster. Information about the effect of the substance which causes a disaster for the first time, will be always insufficient. Diagnosis and evaluation of exposure in the field with the most simple procedures will, however, be the basis of therapeutic success at all levels of medical aid. The limited diagnostic and therapeutic possibilities in disastrous situations must be reflected in the legislation.

Estimate of Exposure in the Area of the Disaster by the Modelled Spread of the Toxic Chemical in the Environment

Mathematical models of the spread of chemicals in above-ground levels of the atmosphere are of fundamental importance for defining *a priori* the area from which people must be evacuated. Construction of such models and their correction are possible solely in connection with methods of exposure estimates, described above.

Possible inaccuracies in the estimate of critical zones of exposure are usually coordinated by the use of suitable safety coefficients and more pessimistic variants of the spread and escape of substances. The primary advantage is a global estimate of the situation and the possibility of simulating the process in advance. Every model of a real phenomenon is always an approximation of reality. Conditions in which the model is valid are usually not as anticipated. To devise models of very complex phenomena, like the spread of chemicals in space, is impossible without simplification.

The present approach to modelling the expansion of chemicals in acute accidents is based either on mathematic-analytical deduction or empiricism. In the first case it is the application of theoretical principles of diffusion, flow, heat propagation, turbulence as well as statistics to the given problem. In the second case it is the utilization of experience gained by analysis of experimental or concrete situations of substance escape (explosion, fire, JET and freezing flow mechanisms, etc) on the basis of which specific predictive functions are formed (Vertkatram, 1985; Hanna, 1985; Bartknecht, 1980; Butcher and Parnell, 1979; Blackshear, 1974).

However, in the first case, too, the choice of required parameters is empirical. More advantageous appear to be models that are less sensitive to inaccuracies and lack of input data, require less complicated calculations, and

do not hide their approximative nature by the formal complexity of the mathematical apparatus.

The primary problem in the construction of models is acceptance of the imagined shape of the space into which the chemical expands. The basic approaches set out from the notion of semiellipsoid, semicone or three-dimensional Gauss' distribution. The situation is close to a slow escape of the pollutants into space (Teverovskij, 1980). The shape and size of the exposure space is similarly affected by meteorological influences, especially the velocity of wind and vertical stratification of the atmosphere. Applicable corrections for the meteorological situation were described by Sutton (1953) and Pasquill (1975). Considerations of rain have not as yet been included in models, in cases of acute escape it serves rather as a reserve for the benignity of the process.

An entirely accurate description of the expansion of chemicals will in real situations remain an unattainable ideal in every model. Witnesses of accidents frequently speak about the non-physical behaviour of chemical clouds. They describe mysterious amoebiform or snake-like formations which transform incalculably and move inexplicably. The movement of clouds can be influenced by slight anomalies in the terrain or by undercooling. The situation is illustrated by the escape of 28 500 litres of liquid ammonia at the bridge crossing in Houston in 1976 (Dorias, 1984).

Not even the escape of the substance from the container need be linear. At a smaller opening of escape expansive cooling may lead to a blockage or periodicity of escape. Explanation of a number of anomalies would be possible on the basis of the theory of dissipative structure or of catastrophe theory (Ebellig, 1976; Poston and Steward, 1978; Thomson, 1982). Experimental modelling and verification of various models for the expansion of heavy gas is excellent in McQuaid and Roebuck (1985); Vilain, (1986); Kirsch and Schnatz, (1985); Riethmuller *et al.* (1985).

A very general approach to the construction of models is the application of the 'law of 2/3 exponent' based on dimension analysis. The procedure is little sensitive to changes in the shape of exposure space. On this principle a very general model was described by Altunin (1978) visualizing the spread of chemicals in semiconical form. Its basis is estimation of the maximum distance the front of the toxic cloud can reach in the above-ground atmospheric layer at critical values of the exposure product for the effect considered. This effect can be expressed by the probability of impairment or death in a defined area. In case of winds the above-ground zone is visualized as the sector of a circle, at calm in the shape of a circle around the epicentre of the escape.

The model takes account of the velocity of wind and of three basic quantified qualities of atmospheric stratification. Of advantage is the fact that a change in the hypothesis about the shape of the noxious area can be expressed solely by a change in the basic model constant. Thus its value can be experimentally corrected. In view of the use of the critical exposure product in the formula,

internal shortening eliminates the time that would have to be considered non-linearly. The model is expressed by the formula:

$$L = B_x \sqrt[3]{\frac{M^2}{C^2 \times V^2 \times K^2}}$$

where L = the depth of contamination in metres
 M = the weight of the chemical in kilograms
 C = the exposure product corresponding to the probability of the chosen effect
 V = wind velocity in metres/second
 K = coefficient of vertical stratification of the atmosphere
 B = the theoretically derived or empirically determined coefficient corresponding to the shape of the exposure area.

In case of calm and inversion the least favourable estimate is calculated as $K = 2$ and wind velocity 1 m s^{-1} in the shape of a circle of L radius. The escape rate of the substance is not defined closer in the model and the notion of propagation in a sector of a circle at movement in the atmosphere is somewhat controversial.

We have attempted to improve the model by introducing correction for the escape rate of the substance, based on the concept of expansive velocity. As the ground surface projection area we chose instead of a sector in a circle an ellipse at whose construction account is taken of wind velocity and expansion. On the basis of the equation of second degree it is possible to calculate the time estimate at which the front of the chemical reaches the edge of the ground surface area.

The model is of an adaptive nature. On fixing the value of coefficient B , it is possible to calculate, on the basis of concrete data the frequency of damage at different distances from the epicentre of the accident, the estimated exposure product for the escaped chemical. The results can be verified by analysis of actual concentrations in the environment. In a similar way the model coefficient B can be made precise by regression analysis.

For a practical application of the model a minicomputer, e.g. type HP 85, will suffice. We use it in our laboratory and it provides numerical and graphical estimates of the endangered area and persons. The model can be used to estimate the size of the evacuation area and the synchronization of time of rescue operations. Calculations are made for the penetration of the toxic agent into defined radial sectors around the site of escape in which correction is automatically performed for basic field influences on expansion.

Going out from the original number of sixteen evacuation sectors we regard as more suitable the system of eight sectors (Thiess, 1979). Every application of the model in a real situation must be verified in retrospect and corrected on the basis of objectively determined concentrations and intensity of exposure in the area of the disaster. It is necessary to make precise not only the basic topological parameter of the model but also to calculate by regression analysis the estimates of exposure products of the escaped substances for determining the impact of key effects on man.

The model of chemical escape is one of the basic elements of accident projects which serve as the basis for rational prevention and rescue measures at a chemical disaster in a plant. Through simulation of different variants of escape as well as the meteorological situation, we can calculate the time characteristics of reaching points of population clusters and select optimal evacuation trajectories. Here the interdisciplinary approach of mathematicians, technicians, chemists and physicians will be effectual in planning cooperation and efficiency of all operations.

On the basis of a mathematical simulation of the most unfavourable variant of the calamity, consisting of the fictitious effect of all the stored toxic substances, it is possible to calculate the maximal toxic potential of the plant expressed in the theoretical number of vitally affected individuals in the action area of the disaster.

6.5 RECOMMENDATIONS

1. For most of the industrial chemicals the values of exposure products and parameters of toxicity for man are not known. From every chemical accident the maximum of relevant information about exposure, effect and efficient therapy at intoxication with the given agent, must be obtained.
2. There are shortcomings in the world literature with respect to documentation of results of toxic exposure in animal experiments. Besides the standard value of LD_{50} , the second parameter of steepness or LD_{16} value must be documented in order to extrapolate the predictive effect to the whole range of doses.
3. To express parameters of toxicity in molar concentrations of the substance theoretically provides no particular advantage; for practical purposes of exposure estimates weight concentrations are preferable.
4. For individual substances it would be well to normalize data about toxicity on an international level, using standard models of the effect of substances.
5. In view of the small frequency of chemical disasters, generalized knowledge can be achieved only by long-term collection and analysis of data on a worldwide scale.
6. It is necessary to devise and verify procedures that will permit the most quantitative determination of exposures and the danger to the individual of intoxication.

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